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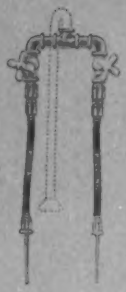

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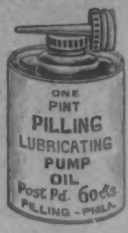

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THE
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VOL. XL. NOVEMBER, 1930. No. 11

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

SOME CONSIDERATIONS AND SUGGESTIONS IN THE HANDLING OF LATERAL SINUS THROMBOSIS.*

DR. A. T. WANAMAKER, Seattle, Wash.

It is taken for granted that every otologist is familiar with the cardinal symptoms and the accepted treatment of lateral sinus thrombosis. However, there have been some problems which the author has tried to solve and some points which he has felt have been helpful to him. These he presents for what they are worth. Having noted the incidence of sinus thrombosis, occurring in the practice of the following authors:

7 cases in 166 mastoid operations by Hill;
5 cases in 79 mastoid operations by Downey;
25 cases in 524 mastoid operations by Garber;
3 cases in 100 mastoid operations by Welty;
19 cases in 497 mastoid operations by Mass. Eye and Ear Inf.;
6 cases in 192 mastoid operations by Naftzger[1];
(26 cases in 487 mastoid operations by Seattle Ear Surgeons);

the question arose as to how often this complication, in the practice of all ear surgeons taken as a group, occurred in a certain locality. To this end, a careful survey of the mastoid records of all the hospitals and public surgeries in the city of Seattle was made for the years 1925, 1926, 1927 and 1928—a period of four years. During this time there were 487 cases of mastoiditis operated upon. Of

*Read before the American Laryngological, Rhinological and Otological Society, May 30, 1930.

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these there were 26 of sinus thrombosis, or 5.75 per cent. Twenty cases occurred in acute ears, six in chronic ears, 16 were on the right side, or 62 per cent; 10 were on the left side, or 38 per cent. It was found to be the most freqent complication. These statistics are rather higher than those reported by the above men. As their reports were made several years ago, it may be that we have been dealing with more virulent ear infections in the past few years. On the other hand, prominent men, such as those reporting their cases, should have a higher percentage of sinus thrombosis to deal with, inasmuch as some of their confreres often refer such cases to them (Fig. 1).

The following diagnostic aids are to be stressed:

The Blood Culture: A blood culture with the first signs of sepsis should be taken, which should be done soon after the chill, if the patient is having any, or just as the temperature starts on its downward course. It has been positive in eight out of nine cases in my practice.

The Leukocyte Count: During the past year, I have been convinced by Dr. A. W. Howe, of Tacoma, of the value of the leukocyte count according to the formula of Arneth and Schilling, inasmuch as it gives an index as to the ability of the blood to handle the infection. Quoting Niehaus[2]: "Certain blood pictures indicate certain phases of disease or infection. *A.* Slight stimulations produce only minor changes with a slight increase of staff forms. *B.* A moderate stimulus shows a few young forms. *C.* Strong excitants cause a marked increase of the young and even parent forms (myelocytes). Extreme stimulation may cause a central injury and a marked disturbance of peripheral cells.

"Ordinarily, the leukocyte count is parallel with the severity of the infection, but with a very grave infection a sudden fall may occur. In the latter instance, the decrease might be interpreted as an omen of recovery, but in reality it would be the reverse. The correct interpretation would be evident by a proper scrutiny of the individual cells. Undoubtedly many young forms would be present in the blood. This phase concerns only the granulocytes. When this ends favorably, with a return of the nuclear deviation to the right and a decrease of leukocytosis, there is an increase of monocytes. This denotes that the infection has been conquered. The healing phase is evidenced by a lymphocytosis. Eosinophils also disappear from the blood in severe infection and their reappearance is the earliest favorable sign. On the other hand, basophils appear only with severe infection, when the defensive factors are losing ground. They again disappear with earliest improvement."

When the leukocyte count becomes low, in the presence of a severe infection, I believe a blood transfusion should be done immediately.

The Urine: The urine should be centrifuged and stained for bacteria. In a few cases streptococci were found in the stained smears of our septic thrombosis cases before we found them in the blood.

X-ray: Stereo X-ray pictures of both mastoids are invaluable, not only in showing the condition of the cellular structure and the size

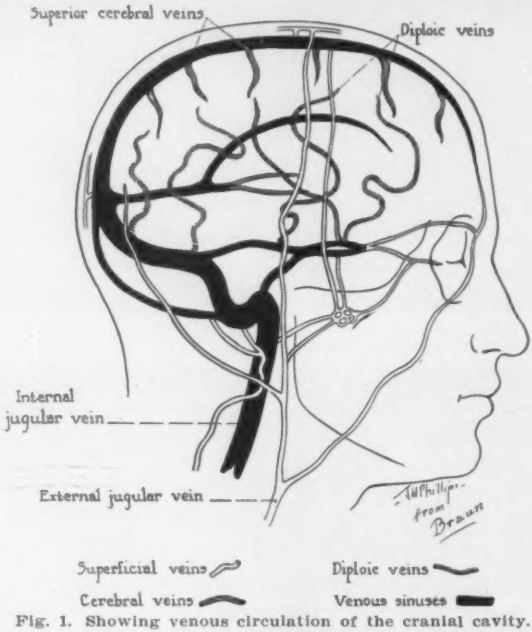

Fig. 1. Showing venous circulation of the cranial cavity.

of the mastoid process, but also the location of the lateral sinus. The posterior anterior position (the occiput down) will very often give a picture of the transverse sinus on each side, which can be compared for size. This comparison is important, inasmuch as anomalies of this sinus are found more commonly than we are given to believe.

Ayer-Tobey[3] or Queckenstedt's sign: Inasmuch as the right lateral sinus usually carries the greater part of the return blood from the cranial cavity and is the side thrombosed in over 60 per cent of cases, it is important in all right-sided infections to perform this

test and thereby note if there is any abnormal reading of intracranial pressure when the left jugular is compressed. Should the left lateral be of normal size, pressure over the left jugular will cause a marked rise in the manometer reading when the right is thrombosed. Should the manometer reading be above normal without any jugular compression and change very little when the left jugular is compressed, one should suspect a much narrowed or absent lateral on that side.

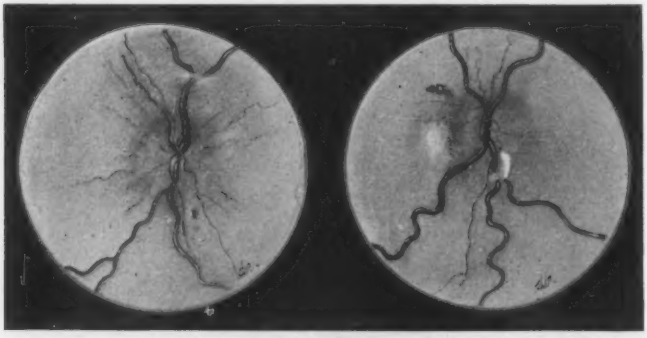

Fig. 2. Bilateral choked disc following ligation of the right internal jugular vein in a lateral sinus thrombosis.

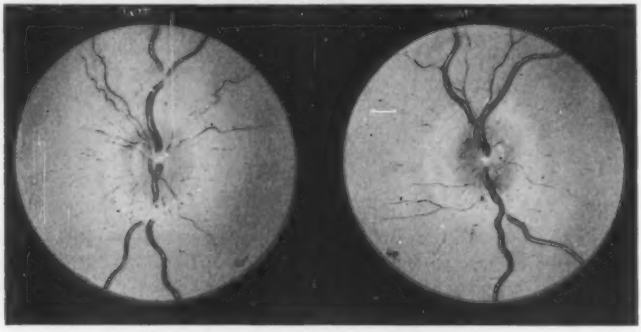

Fig. 3. Drawing made two months later.

When both mastoids are involved, this test is of great value in determining the thrombosed side. Under certain conditions this test is subject to error[4 5].

According to E. Blair Sutphen[6] the changes in the eye grounds are apparent in one out of every five cases. They vary from a slight fullness of the retinal veins upon the diseased side to a real neuroretinitis of both sides, but are more marked upon the diseased side.

This occurrence has also been noted after ligation of the jugular vein and when the operation demonstrated an occluding clot in the sinus, and caused him to wonder if the eye ground choking did not develop because the petrosal circulation was cut off.

The finding of a choked disc in the presence of a sinus thrombosis used to cause me a great deal of worry, in that I felt there was some other intracranial complication present. Now we know that this should be no cause for alarm.

Dr. Goldman and Dr. Maybaum[7], in a report of nine cases of jugular bulb thrombosis, had two cases with a papilledema, in both

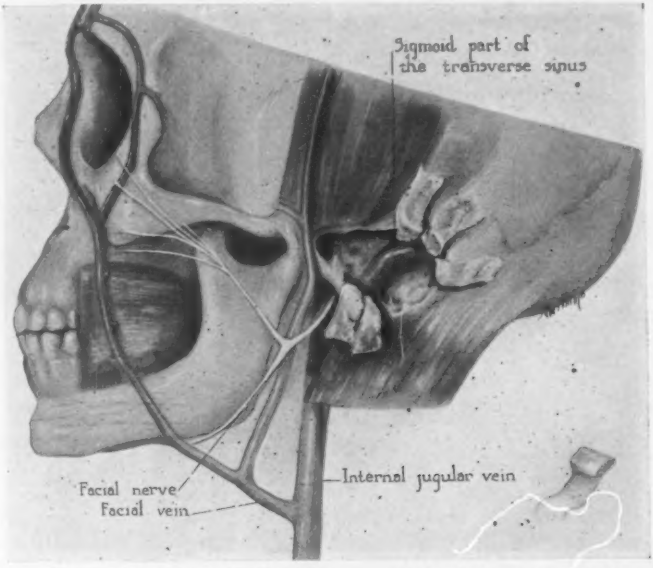

Fig. 4. Sinus exposed and where compression plugs are placed.

of which the swelling in the disc gradually became normal after operation. Dr. Jesberg[8] reported a case of choked disc in a case of mastoiditis with extradural abscess located under the knee of the sigmoid sinus. Dr. H. I. Lille[9] had four cases of choked disc associated with mastoid disease without an apparent intradural involvement. Two showed perisinus abscesses, one a large cholesteatoma with pressure on the sigmoid, and in one he was unable to determine the cause. Ginsberg[10] had a case of bilateral choked disc following the ligation and shutting off of the lateral sinus, which gradually subsided in a little over six weeks.

In recent sinus thrombosis cases, I have studied the eye grounds before and after ligating the jugular vein. In 12 hours there is noted a mild congestion of the disc, which subsides in a day or two. In one case due to continued chills, the jugular was ligated four days after the lateral sinus was shut off. Following the ligation, a choked disc of 3 diopeters with small retinal hemorrhages in each eye occurred (Fig. 2). It is one year since this was done and the fundus is now normal (Fig. 3). In this case, I felt there was some anomalous condition present in the sinus of the opposite side. A study of a stereo X-ray showed the left or sound lateral sinus much narrowed.

My observations are in accord with those of Dr. Swift[11], who believes that congestion and choked disc are due to interference

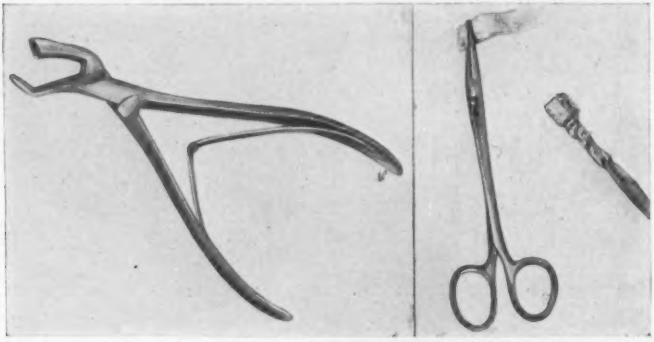

Fig. 5. (a) Lombard's forceps for removing the hard sinus plate without injuring the wall. (b) Method of making compression plugs.

with the return of venous blood from the cranial cavity. This may be due to pressure of a brain abscess or tumor on the transverse sinus wall, a perisinus abscess over the sigmoid or a clot in the sinus, particularly in the jugular bulb, thus damming the circulation in the cavernous sinus where there is absence or anomaly of the other lateral sinus. In consideration of this theory a study of the embryological development of the sinuses is instructive.

ANATOMICAL CONSIDERATIONS.

A mental picture of the junction of the superior petrosal sinus is very useful. This can be gained only by dissection. Occasionally it will be found very difficult to stop the bleeding above, but familiarity with this anatomical location makes the task much easier. Compression plugs made by wrapping one-half-inch iodoform gauze around

a hemostat, so that the inside end may be drawn out. permits one gradually to withdraw pressure without removing the whole plug. This one frequently does on the fifth or sixth day (Figs. 4, 5, 6 and 7).

Braun[12], in his textbook, has illustrated two type of jugular bulbs. In the whole mastoid where the sigmoid sinus is well back, there is a very low vault, while in the narrow mastoid where the sinus is close to the mastoid antrum, the jugular vault is high and may not be reached by a curette (Fig. 8).

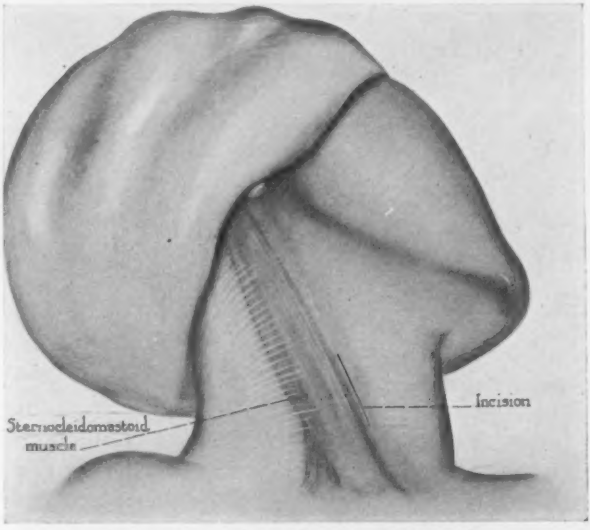

Fig. 6. Size and location of usual incision.

My associate, Dr. Fleischhauer, has under his care at the present time a boy whom he was called to see on account of spontaneous hemorrhage from the middle ear. The hemorrhage was so severe that it was necessary to perform a simple mastoid operation and shut off the lateral sinus; at the same time, ligating the jugular in the neck. The sinus in this case did not approach close to the mastoid antrum, yet the bulb reached up into the middle ear.

Experimental Data: In 1914, the author read a paper on Lateral Sinus Thrombosis with a report of five cases, before the Pacific Coast Ophthalmological and Otolaryngological Society, in which he advised the ligation without severing, if the vein were free from

clot, of the jugular above the entrance of the facial vein, assuming that Nature would eventually re-establish the lumen. In order to test this belief, experimental work was undertaken on dogs. The right internal jugulars of two dogs were tied a half-inch apart with chromicized catgut ligatures. Five months later, these veins were removed, injected with sodium iodid solution, and X-ray photographs were taken. The slide shows clearly the re-establishment of the lumen in the vein of the first dog. The removed specimens were examined by D. H. Nickson, pathologist to the Swedish Hospital, whose report is as follows:

"Specimens consist of a section of the jugular vein 6 c.m. long. A probe could be passed through the lumen with ease. Twenty per cent solution of potassium iodid was injected into the lumen and

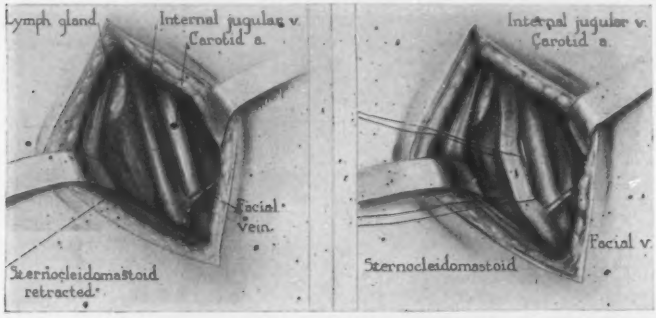

Fig. 7. (a) Vessels exposed. (b) Method of ligating after determining that free blood is present in the vein.

X-ray pictures taken, which show the solution throughout the length of the veins, thus proving that the lumen is intact throughout."

In 1927, a case was observed where the jugular vein had been ligated and the sigmoid sinus shut off by compression plugs some three months before. An operation was undertaken to clean out cells that were still infected. To our surprise, we opened directly into an intact sigmoid sinus which bled so profusely that it became necessary to shut it off above and below. Why do this, when there is a possibility of Nature's closing the cut wall and re-establishing its channel?

Dr. Hans Brunner[13], of Vienna, claims to have a specimen of a recent sinus thrombosis, taken at postmortem, which shows the change of the thrombotic material into granulation tissue. He states that autopsies have shown many small channels making their way through an old thrombosed cavity. It would be very interesting to

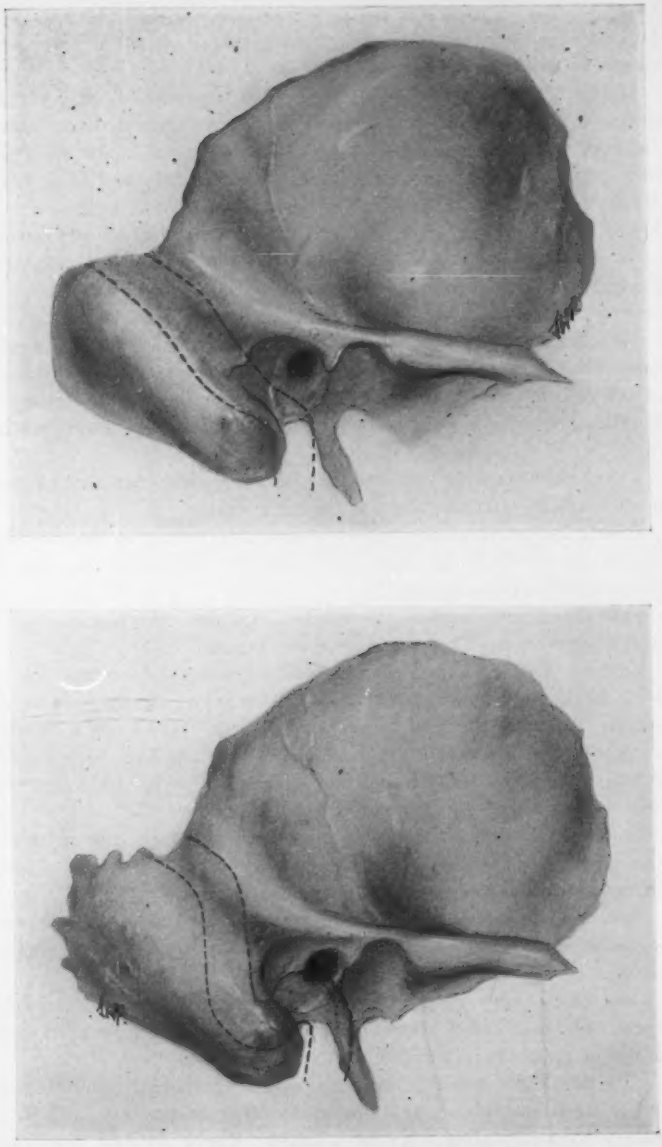

Fig. 8. Illustrating two types of jugular bulbs.

have autopsy reports on the condition of the lateral sinus and jugular vein on cases which had been operated on some years previous for sinus thrombosis.

Heidrich[14], in a summary of cases over a period of 10 years, in which various vessels were ligated, says that cerebral disturbances followed in 30 per cent of 53 common carotid cases ligated; 4.4 per cent of 23 cases in which the jugular was ligated; and in another series 7.1 per cent of 14 jugular vein cases. No disturbance followed when vertebral arteries were ligated, or in 21 cases of external carotids. He states that thrombosis allows a change for collateral circulation. Later thrombosis may allow the passage of blood, restoring the permeability of the blood vessels.

Case Reports (see Table I): The following is a report on twenty cases of lateral sinus thrombosis and two cases of blood stream infection complicating mastoiditis; one case of jugular phlebitis and jugular bulb thrombosis, complicating suppurating glands of the neck.

Blood cultures were not made on the early cases nor on two that were operated on for colleagues in other cities.

Case 2 had a complete jugular phlebitis extending to the clavicle. There was no discharge from the middle ear. Infection was due to the streptococcus mucosus.

Case 6 had no symptoms of sepsis. Organized thrombosis discovered at operation. The jugular was not ligated.

Case 12 began a septic temperature after double mastoid operation. Both lateral sinuses were exposed. Slight bleeding of sinus wall on removing the sinus plate on right side. After one week the sinus wall did not take on the healthy granulations like the left side. It was, therefore, decided to ligate the right jugular and open the right lateral sinus, which showed a mural clot.

Case 16 had a bronchial pneumonia complicated by double mastoiditis. The left or first ear to drain showed a grayish, doughy wall through which no circulating fluid could be felt.

Case 21 had a perisinus abscess with marked septic symptoms, headache and diarrhea. The recovery was prompt after the simple mastoid operation.

Case 22. Double mastoid. Ran a septic temperature for several weeks. She also had a hemorrhagic nephritis. Her temperature and condition improved immediately after transfusion.

Case 23. Eight months Japanese baby with jugular phlebitis and lateral sinus thrombosis and pneumonic area in the left lung, secondary to suppurating cervical adenitis. There was no operation but an autopsy was performed.

Name	*Year*	*Side*	*Acute*	*Age*	*Culture from Mastoid*	*Blood Culture*	*Sinus Shut Off*	*Jugular Ligated*	*Fundus*	*Transfusion*	*Complication*
1. Miss T. D.	1912	R	Yes	22	Staphylococcus	Not Taken	Yes	Yes			
2. Ruth H.	1912	L	Yes	6	Strep. Mucosus	Not Taken	Yes	No			
3. Peter P.	1913	R	Yes	25	Not Taken	Not Taken	No	Yes			Abscess chest
4. H. R.	1913	L	Yes	21	Streptococcus	Not Taken	Yes	Yes			
5. Ivan W.	1914	L	Yes	6	Staphylococcus	Not Taken	Yes	Yes			
6. Mrs. S.	1914	R	Yes	50	Not Taken	Not Taken	Yes	No			
7. John G.	1915	R	Yes	6	Streptococcus	Not Taken	Yes	Yes			
8. Aberdeen Case*	1916	R	Yes	7	Not Taken	Not Taken	Yes	Yes			
9. Mr. T.	1922	R	Yes	38	Streptococcus	Not Taken	Yes	Yes			
10. Spokane Case	1924	L	Yes	25	Not Taken	Not Taken	Yes	Yes			
11. John R.*	1925	R	Yes	6	Streptococcus	Not Taken	Yes	Yes	Slight choke	Yes	Brain abscess
12. Temple W.	1025	R	Yes	7	Strep. Hemo.	Positive	Yes	Yes			
13. Vivian Z.	1925	L	Yes	7	Strep. Capsul	Positive	Yes	Yes			
14. Jane T.	1926	L	Yes	10	Diplococcus	Not Taken	Yes	Yes			
15. Ann S.	1925	R	Yes	7	Streptococcus	Negative	Yes	Yes		Yes	
16. Emmy Lou B.	1927	L	Yes	4	Streptococcus	Not Taken	Yes	No			
17. V. Olson	1927	R	Yes	9	Strep. Non-hemo.	Positive	Yes	Yes			
18. V. Nelson	1928	L	Yes	15	Strep. Hemo.	Positive	Yes	Yes	Choke after ligation	Yes	Elbow joint gluteal abscess
19. Helen C.	1929	R	Yes	6	Strep. Hemo.	Positive	Yes	Yes			
20. Tacoma Case*	1929	R	Yes	42	Strep. Hemo.	Positive	Yes	Yes			
21. Mortno S.	1916		Yes	12	Streptococcus	Not Taken	No	No			
22. Mrs. B.	1923	R	Yes	35	Streptococcus	Postive	No	No		Yes	
23. Baby Takai*	1926	L	Yes	8 mo.	†		No	No			

*Died.

†Jugular phlebitis and sinus thrombosis secondary to suppurating glands of the neck, 20 cases operated upon—85 per cent cured.

Last July, I was able to check 11 of these operative cases. All were in splendid physical and mental condition. One, a boy operated in 1915, is stroke on the University of Washington varsity rowing crew.

THE CONCLUSIONS ARE:

1. Lateral sinus thrombosis occurs more commonly than reports indicate.

2. Von Schilling's method of leukocyte count is useful for prognosis and treatment.

3. Large numbers of streptococci may be found in the urine before they are found in the blood.

4. Choked disc is the result of mechanical obstruction.

5. Stereo X-rays are of value, not only in determining the condition of the mastoid process but also for locating the position and size of the sigmoid sinus on both sides.

6. The Queckenstedt or Ayer-Toby test is useful in diagnosing a mural clot, an obliterating clot or a narrow or absent lateral sinus on the non-infected side.

7. Where possible the sigmoid sinus wall should be preserved.

8. Experimental work on dogs has shown the value of not severing the internal jugular vein.

9. A lateral sinus operation does not weaken a patient physically or mentally.

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A METHOD FOR THE REPAIR OF PERSISTENT POST-AURICULAR OPENINGS.*

DR. RALPH ALMOUR, New York.

The method herein described is for use in those cases following simple mastoidectomy wherein a large bony defect persists and the skin wound does not heal. Every otologist has seen cases following simple mastoid operations where for one reason or another the mastoid cavity does not fill in with connective tissue and the overlying skin wound does not heal. Such cases, when seen clinically, show a large opening in the scar over the mastoid process and a large cavity

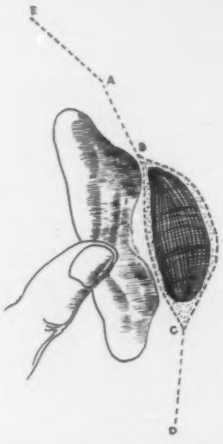

Fig. 1. A method for the repair of persistent postauricular openings.

within the mastoid process which practically corresponds to the originally exenterated mastoid.

I have found that it is not sufficient, in these cases, to curette the wound and suture the periosteum and skin. Following this procedure, there always remained a hollow underneath the skin, in which serum and mucus would accumulate. After several unsuccessful attempts, I have employed the following method with marked success in four cases.†

*From the Otolaryngological Department of Beth Israel Hospital.

†Using this method, I have operated on three cases with successful outcome. In two of these, previous attempts at postauricular closure by the usual methods were unsuccessful.—S. J. Kopetzky.

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Technique: The incision is started at the upper attachment of the auricle, about one-fourth of an inch back, and carried through the old scar down to the edge of the opening (Fig. 1, A-B). The opening is then circumscribed by an incision placed at either side of it (Fig. 1, B-C). These two incisions meet the first incision above and join each other below. The scar around the opening is then excised and the skin and periosteum retracted forward and backward. From the upper angle of the first incision another is extended at an angle of 45 degrees into the scalp above the hairline (Fig. 1, A-E). This incision involves only the skin and extends down only to the superficial fascia covering the temporal muscle. An area of muscle which

Fig. 2. A method for the repair of persistent postauricular openings.

will fill the mastoid cavity is roughly outlined by an incision so that the pedicle of the muscle is situated at the upper anterior angle of the mastoid cavity (Fig. 2, A). The incision is then carried down to the bone and periosteum. The area of muscle already described is circumscribed by this incision, separated away from the underlying bone and swung into the mastoid cavity so that it is attached to the remainder of the muscle by a narrow pedicle located in the upper anterior quadrant of the mastoid cavity (Fig. 3, A). This point usually corresponds to the angle formed by the zygoma and superior canal wall. The muscle is placed in the mastoid cavity so that its periosteal surface faces externally and its former external surface

faces the bony walls of the mastoid cavity (Fig. 3, B). A suture is then taken so that the middle of this muscle flap is tied to the lower portion of the under surface of the anterior flap of the skin wound. Another suture is taken so that the lower portion of the muscle is attached to the lower angle of the under surface of the skin wound. The skin and periosteum, after having been refreshened, are then brought together by interrupted mattress sutures of either silkworm or tension. These are inserted through the skin and periosteum and then brought back through the edges of the skin wound only. In this way the superficial wound is brought together over the muscle embedded in the mastoid cavity and a firm wound results.

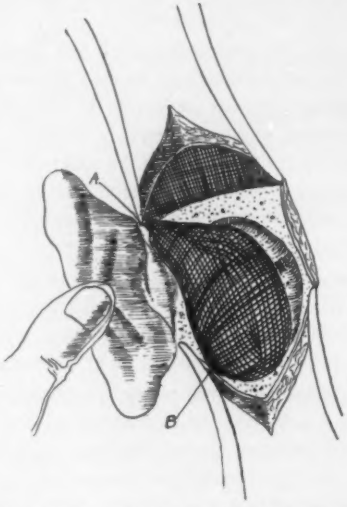

Fig. 3. A method for the repair of persistent postauricular openings.

For approximately three or four weeks after primary union has resulted there is noted a slight bulging of the wound, due to the excess of muscle tissue in the cavity. This, however, soon flattens out as the muscle attaches itself to the bone and undergoes fibrotic contraction. This method is not to be employed where any suppuration is present in the old mastoid cavity. It is to be used only in cases where no infection is present, for infection will cause a breaking down of the entire superficial wound and a sloughing out of the muscle pedicle.

51 West 73rd Street.

AUDITORY NERVE DEAFNESS.*

DR. A. SCHATTNER, New York.

In presenting this paper to you this evening, I am conscious of the fact that the term Auditory Nerve Deafness embraces innumerable clinical entities and it shall be my endeavor to present this subject in as clear and concise a manner as possible, and at the same time not to bore you too much with details. The etiology and the clinical manifestations only shall be dealt with, the therapeutic aspect being beyond the scope of this presentation.

Before going into the real subject of the paper let me mention certain anatomical and physiological factors which have a direct bearing upon the clinical pictures presented by disease of the auditory apparatus. The name auditory nerve is a misnomer, but has been kept in the medical literature because the main function of the eighth nerve is auditory, the vestibular division having been discovered at a later date, at which time the term auditory had already become popular and synonymous with the eighth nerve.

From a clinical standpoint we should speak of auditory and vestibular nerve apparatus, since it is usually impossible clinically to differentiate lesions in the end-organs and the nerve proper; moreover, the lesions frequently involve both at the same time.

The very complicated auditory nerve apparatus consists of at least five neurons, there still being disagreement among the investigators as to the exact course taken, so we shall not trace its central course. Suffice it to say that both rami cochleares distribute fibres to each temporal lobe. Disturbances of hearing of a high degree are not induced by diseases of the cortex.

The relations of the ramus vestibularis are not so complicated. The vestibular nerve apparatus consists of three neurons. Deiter's nucleus is now looked upon as a nucleus of co-ordination, having relations on the one hand to the cerebellum and the vestibular nerve, and on the other to the nerves of the muscles of the eye and of the cervical muscles, explaining clearly the important role it plays in reference to the perception of our position in space and to the occurrence of nystagmus.

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Politzer brought to our notice in considering the frequency of disturbances of hearing, that the auditory nerve possesses the greatest impressionability of all the cranial nerves, that is, its function is much more often arrested by general disease and chemical changes in the blood during infectious diseases than that of the optic, gustatory, olfactory and sensory nerves.

Alexander offers the following explanation for this very important phenomenon:

1. In the membranous internal ear two organs of very different phylogenetic age are paired. The labyrinth proper, that is, the utricle and semicircular canals, takes its origin deep down in the animal kingdom, even nonvertebrates possessing a labyrinth called statocyst; furthermore, some plants reveal cellular organs which are homologous to the statocyst.

The cochlea appears much later and is first found in amphibia, reptiles, birds and mammalia.

It is an established fact that the resistance of an organ increases with its phylogenetic age and, therefore, it is easily understood, that the same congenital injury may leave the labyrinth unchanged, whereas the cochlea may be impeded in its development.

2. The second factor of vital importance is revealed by the histological findings of the end-organs. The auditory end-organ consists of much smaller and more delicate cellular elements and nerve fibres than does the end-organ of the vestibular nerve. The resistance of an organ to injury is decreased in proportion to the delicacy of its cellular elements.

The great majority of cases of acquired deafness may be traced anatomically to an inflammation of the internal ear. The etiological factor in these cases is so severe and its influence so marked that usually the whole internal ear (cochlear and vestibular apparatus) is destroyed. Therefore, typical findings of acquired deafness are: total deafness without rests of hearing and nonirritability of the vestibular apparatus.

The typical findings of congenital deafness are, on the contrary, deafness with functioning vestibular apparatus. In a great proportion of these latter cases rests of hearing remain.

Disease of the internal ear are met with more often in children than in adults. This is explained by the fact that diseases, such as the acute exanthemata, diphtheria, acute hydrocephalus, epidemic cerebrospinal meningitis, etc., which occur so frequently in children are often associated with affections of the ear. Moreover, it must be pointed out that the anatomical connections between the middle

ear, labyrinth and cranial cavity are more numerous in children than in adults and, further, that by means of aqueducts there is a more extensive communication between the fluids of the labyrinth and the cerebrospinal space in children than in adults.

The frequency of affections of the auditory nerve diminishes in middle age, but increases again considerably in the later years of life, even after the sixtieth year. The anatomical causes of this disturbance of hearing in old age, in addition to changes in the labyrinthine capsule which lead to ankylosis of the stapes (otosclerosis), are due to retrograde changes in the auditory nerve and its terminations (senile degeneration), such as atrophy, fatty degeneration and chronic endarteritis.

Statistics of affections of the auditory nerve are sparse and unreliable. That these affections occur more frequently in males may be explained by the fact that they are more exposed to harmful influences of certain occupations. Both ears are usually affected.

Alexander states that in his experience the percentage of congenital deafness is slightly less than that of the acquired form. This, however, varies greatly in different regions, where factors such as endemic cretinism come into play.

The present classification of auditory nerve deafness is unsatisfactory, because different investigators have classified these affections upon different bases, such as etiology, anatomy, pathology, congenital and acquired, etc. For clinical purposes I have accepted Alexander's new classification, in which he combines clinical-anatomical findings.

Alexander divides the diseases of the internal ear and eighth nerve into three main groups, each of which is subdivided:

I. The first main group comprises *constitutional, hereditary, degenerative affections of the internal ear and eighth nerve.* This is subdivided into *a.* fetal and *b.* postfetal, to which latter belong *1.* the juvenile form of progressive internal ear disease, and *2.* otosclerosis.

I shall not dwell upon this group as it would take too much time, and we now turn to the second Group II, *individually acquired affections of the internal ear and eighth nerve,* with which we shall now occupy ourselves. This group is again subdivided into: *a.* Fetal, caused by trauma, lues and acute infections. *b.* Postfetal following *1.* trauma. Here a variety of clinical pictures present themselves.

Mechanical trauma may be due to the action of intense sounds (explosions, detonations), indirect injury to the labyrinth, injuries to the skull (fall on the head, fall, blow and concussion) and injuries to the labyrinth occasioned by sudden changes of air pressure, as in

caisson workers, aeronauts and divers. The majority of aural affections in caisson workers occur during the stage of decompression, if this is carried out too rapidly. Without going into a description of the tympanic lesions it is important to note that gas emboli, due to the development of gas in the blood, may cause permanent disturbances of hearing through ischemia, and local destruction of the central fibres of the auditory tract, of the auditory nerve and of its branches. We must also add here labyrinthine disturbances in hearing which are caused in persons who have certain occupations, as blacksmiths, locksmiths, plate workers, coppersmiths, coopers, engineers, stokers. Temporary or permanent disturbances of hearing following certain affections of the mind are explainable upon the basis of the vasomotor nerve mechanism.

That the auditory nerve is more often affected than any of the other nerves of special sense, is clearly demonstrated by the action of drugs circulating in the blood, producing chemical trauma. One must only take into consideration the action of such medicaments as quinin, salicylic acid, morphin, chloroform, tobacco, alcohol and the oil of chenopodium, which exercise a temporary, often however, a permanent influence on the functions of the auditory nerve, inasmuch as they cause subjective noises and deafness, rarely disturbances of vision, when used in large doses for a long time. Other toxins having a paralyzing effect on the auditory nerve are lead, mercury, arsenic, carbon disulphid, anilin preparations and phosphorous. Metabolic disturbances and deficiency of vitamins may also be considered in this class.

2. Meningitis is the next class of cases to be considered. That due to the meningococcus arouses our special interest. The other meningitides, such as the tuberculous, the latent form in congenital syphilis, traumatism following fracture of the petrous bone, etc., reveal such a typical clinical picture that we just wish to call your attention to them. Meningococcus meningitis, according to some investigators, is responsible for one-third of all the cases of acquired deafness or deaf mutism. The percentage of postmeningitic auditory lesions varies in different epidemics from 2 to 24 per cent. In the majority of these lesions both auditory and vestibular responses are lost completely. In accordance with our previous statements, disturbances of the auditory function outnumber those of visual functions as 8 to 1, and they are also the most frequent of all cranial lesions attributable to meningitis.

The process by which the eighth nerve is involved is usually a direct invasion along the nerve sheaths and dura through the internal

auditory meatus with rapid spread to the end-organs, leading to their destruction.

The neuroepithelium may be destroyed within a few hours from the time of invasion of the internal ear. Almost without exception both ears are involved (91.8 per cent, Bezold). The onset of ear complication is usually in the first week of the disease and is frequently masked by the seriousness of the other symptoms present. From what has been said it is clear that improvement may only be expected in exceptional cases, where mild changes have taken place.

3. The third division of this group comprises other acute infections and is responsible for the majority of cases of acquired nerve deafness. Here the process in the internal ear is usually secondary to a tympanic lesion and a complication thereof. The pathological process may range from a serous to a diffuse purulent labyrinthitis. Where the internal ear is invaded by metastasis, the anatomical changes in the auditory nerve apparatus in the course of these infectious diseases are not yet well understood. Hyperemia and ecchymosis, small-cell infiltration and suppurative inflammation of the labyrinth, with its termination in the new formation of connective tissue and bone, have been most frequently found. The cases of complete deafness following mumps, of which a great number of clinical observations are now on record, are probably due to changes such as were just described.

4. We next come to the fourth, *chronic infections,* which include cases of focus of infection and syphilis. A reasonable assumption based on clinical experience is the basis on which a diagnosis of toxic neuritis of the eighth nerve from focal infection in the tonsils, teeth and elsewhere in the body is made. These functional disturbances of the internal ear sometimes become permanent. Specific involvement of the eighth nerve more often appears as a later form of syphilis, generally at the end of the secondary, or at the beginning of the tertiary stage, or even later. The anatomical changes are but little known. One ear is generally more affected than the other. The course of syphilis of the labyrinth is characterized by the rapid development of disturbances of hearing, often remaining stationary for some time, until, after weeks or months, a sudden change for the worse takes place.

The fifth subdivision of this class concerns rickets. Up to the present time we possess only a few observations as to the changes in the labyrinth in this disease.

III. The third large group is the group of internal ear, eighth nerve affections, following intercurrent diseases in individuals with

a degenerative status. This group is subdivided as the previous group. Here lowered resistance of the auditory organ is the principal factor, whereby the labyrinth is more easily injured by disease and external influences.

In conclusion, I wish to point out that in the preparation of this paper I was guided in my selection of clinical conditions by the frequency with which they are encountered in our clinics. The empirical choice of these conditions was predicated by the above principle. The paper is necessarily incomplete, in view of the great variety of conditions that may be met with in diseases of the auditory nerve and internal ear.

308 West 48th Street.

FATAL IDIOPATHIC PHARYNGEAL HEMORRHAGE. A CASE REPORT.*†

DR. EDWARD RUSSELL ROBERTS, Bridgeport, Conn.

Patient: A. L. History: Nov. 12, 1928. C. C.: Bleeding from the right ear.

P. I.: The mother states that the baby started to have a swelling in the right side of the neck four weeks ago and it appeared to have pain in the right ear. Shortly after the onset, mucopurulent discharge appeared from the right ear. Three weeks ago the patient suddenly had a severe hemorrhage from the right ear, which lasted 10 minutes. This stopped spontaneously but the baby continued to have discharge from the right ear and appeared listless. On three other occasions the patient has had severe hemorrhages similar to those mentioned above. The last attack was yesterday, on which day Dr. Smyth opened a right retropharyngeal abscess and ordered the baby to the hospital.

P. H.: Whooping cough two months ago.

Essentials in Physical Examination: The patient is a pale, listless baby, age 2 years, appearing very anemic.

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*Notes taken from records of the Bridgeport Hospital, Bridgeport, Conn.

†Read before Eye, Ear, Nose and Throat Section at the fall meeting of the Connecticut State Medical Society, Racebrook Country Club, Nov. 14, 1929.

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Ears: Right ear shows purulent discharge of a chronic suppurative otitis.

Mouth: Tongue is coated. Pharynx filled with purulent material, incision of right posteriolateral wall.

Neck: On the right side of the neck on the anterior aspect there is a mass which is firm and circumscribed.

Heart: Rate is 160. Rhythm regular. Sound, a fair quality.

Blood Picture: Hemoglobin, 30 per cent. White blood count, 17,000. Red blood count, 1,700,000. Polymorph., 80 per cent. Blood is Type 4. Swelling on the right side of the neck. Reopening of retropharyngeal swelling showed old bloodclot. During this operation venous blood of the same color presented in the right auditory canal with clots to be seen in tympanum through perforation.

Nov. 21, 1928: Within nine days, aided by transfusion and Alpine treatments, hemoglobin reached 57 per cent, with steadying improvement in the general condition and recession of the right cervical swelling, external and retropharyngeal swelling reduced to a firm oval prominence, 1 inch in its longest dimension.

Dec. 5: Right tympanum dry.

Dec. 12: Hemorrhages from the right ear controlled only by tight pressure bandage, reducing hemoglobin to 52 per cent.

Dec. 13: Blood transfusion.

Dec. 20: X-ray examination of cervical spine shows no evidence of bone pathology.

Pharyngeal swelling embarrassing respirations. Small opening made in it with the tip of a small knife, resulting in a tremendous gush of arterial blood. Rushed to the operating room, where an external carotid artery and internal jugular vein were ligated.

A gauze sponge and sponge forceps were held against the pharyngeal mass during this procedure, during which time several crepitating, sausage-shaped masses, 2 to 3 inches in length and ½ to ¾ of an inch in diameter, presented at the mouth. Presumably these tissue masses were expelled through the very small opening, then the masses themselves.

Histologically, this material was broken down lymphatic structure.

Dec. 24: Pharyngeal swelling larger than ever. Swallowing impossible and suffocation seems imminent. Aspiration with a medium needle attached to a Luer syringe resulted in a violent gush of arterial blood and death followed in a few seconds. The actual bleeding was 22 ounces.

144 Golden Hill Street.

HOW SHALL WE EVALUATE THE VARIOUS MANIFESTATIONS OF ACUTE MASTOIDITIS?*

DR. BEN L. BRYANT, Cincinnati.

Otolaryngology as a constituent tile in the mosaic of medicine has taken part in the developmental changes and vacillations which medicine, as a whole, has undergone since the inception of our specialty. In addition to these, otolaryngology has had a more or less independent transition of its own: a transition from that period of early enthusiasm, which, not unlike the rush to hitherto unexplored fertile territories geographically, attracted to its realm many individuals fired with enthusiasm over the possibilities of a field of medicine likewise unexplored. The development of this special field has been comparatively recent and quite rapid. The early years saw a steady march forward, particularly in operative technique; but the danger during this first period lay in the tendency to emphasize more the how, rather than the why, where or when of the operation. The pendulum, however—true to form—is swinging, albeit a bit ponderously at times, backward; and the observation has been repeatedly made recently that the "specialist" in the old sense must give way to the physician who specializes. It becomes increasingly apparent that the necessity for not only a wider outlook, but a wider use of fundamental anatomical, physiological and pathological facts is necessary- not only in justice to the patient, but also in justice to ourselves as physicians.

How shall we evaluate the various manifestations of acute mastoiditis? We not only shall evaluate them, but we can only evaluate them on a basis of a thorough understanding and careful consideration of the anatomy, pathology, bacteriology, Roentgenology, hematology and immunity entering into and controlling the clinical condition known as acute mastoiditis. No more than an architect could evolve, design and plan a magnificent turret to top and grace an imposing edifice without considering the structural capacity of the foundation and the possibilities of the materials to be used, can we formulate our idea of diagnosis and therapy of this disease picture, without a true conception of the fundamental biological facts underlying it and the changes which have taken place as an expression of the irritating factors. An orderly, painstaking study of divers fac-

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tors is, therefore, absolutely essential. It must be apparent at the outset that diagnostic evidence and operative indication today is not that of 10 years ago, not only because clinical experience has improved clinical judgment, but also because painstaking and patient investigation from scores of faithful, tireless workers during this period has materially amplified the available facts. Of course, I do not mean to infer that the work of the pioneers in this field, who paved the road through the wilderness that their followers might progress more smoothly and intelligently was in vain. On the contrary, many of their maxims have remained as such. A few of their assumptions were erroneous or insufficiently interpreted.

In general, there are three types of acute mastoid involvement: the hemorrhagic or thrombophlebitic, the coalescent and the necrotic; and it is imperative that this primary classification be kept in mind, particularly in establishing an indication for operation. We have just as little sympathy for the otologic surgeon who operates on every case early, as for the one who operates on every case late. It will be our endeavor to assist in clarifying the maze of indecision which often accompanies a consideration of this most crucial point.

That there are widely varying anatomical conditions existing in the mastoid process is an established fact. The final explanation of this is not so well established. Wittmaack and his school have, in recent years, made most valuable contributions to this question and have sought to explain the varying degrees of pneumatization on the basis of middle ear suppuration and mastoid involvement in infancy and early childhood. Their work has been tested by many other investigators and, although certain objections have been raised, their claims have many supporters. It would seem that for the present a safe viewpoint would be that expressed by Knick and Witte, who, after 13 to 14 years, made detailed X-ray examinations of 25 patients who had suffered from otitis media in infancy. They found that of these 25 patients, eight had a definite restriction of pneumatization of the type described by Wittmaack, eight others had no demonstrable disturbance, and the remaining nine were questionable. The conclusion drawn from this work was that otitis media in infancy can restrict the normal pneumatization of the mastoid process, but that it does not always do so; and that the indefinable "individual characteristic" plays a certain role. At any rate, there are variations, ranging from the sclerotic through the diploic and irregularly pneumatized to the extensively, ideally, regularly pneumatized mastoid process. The two mastoid processes of one individual frequently vary, and it is easy to see how this can be explained by the Wittmaack postulate. It is an interesting observation, in passing, that

although the two mastoid processes of an individual may vary, if they are both of the pneumatic type they are practically always nearly identical.

The question of the manner in which the inflammation of the mastoid arises and progresses is one that has been in the limelight of research and discussion in the last several years. Decades ago it was stated that every inflammation of the middle ear had a concomitant inflammation of the mastoid process; and, considering the anatomical fact that the tympanic cavity, the additus, the antrum and the connected cells of the mastoid form one system with continuous lining mucous membrane, it would seem logical that this were true. The question which immediately presents itself is: Why does one suppurating inflammation of the middle ear proceed, in spite of every therapeutic effort, to a mastoiditis requiring subsequent operation, while the next otitis media, with just as stormy a beginning and apparently the same type of mastoid anatomically, as demonstrated by the Roentgenogram, clears up rapidly? Of course, the constitution of the individual, whatever this all-encompassing term can and does include, has a tremendous influence on the outcome. It is a time-proven observation that some individuals react poorly to any type of infection, the striking example of this being the child who has an exudative diathesis. The resistance of the organism, as a whole, has a wide variation, and there is probably no stage to which the inflammation of the mastoid has advanced, short of extensive bony necrosis, which is unable to heal spontaneously, as proven by reported cases. Let me emphasize that I say "which *is unable to* heal spontaneously," the important thing being for us, as physicians, to be so cognizant of the many warning signs and so in command of the various diagnostic aids that we may determine whether the particular mastoid before us has reached the stage where it is irreparable without surgical intervention.

In order to explain the progress of the disease from its incipient to its manifest stages, there has been an increasing and a wise tendency to consider the pathology of the mastoid in the light of general pathological anatomy and, as a definition, we might state that mastoiditis is present in a pathological anatomical sense when, in the course of a middle ear disease, the vascular-connective-tissue apparatus of the mastoid exhibits an increased function as the result of inflammatory irritation. It is a defensive form of inflammation that the mastoid presents, and it is characterized by edema of the tissues, exudation, emigration of blood cells, production of granulation tissue and vascular buds, formation of osteoblasts and osteoclasts, and development of bone-resorbing properties of the endothelium of the

vessels. There is concomitant bone formation and bone destruction. For years there has been a vigorous combat between the school which held that extension of the disease in the mastoid was primarily along the lining mucous membranes of the pneumatic cells and the school which held that it was fundamentally a bone marrow infection. It seems strange that these two viewpoints could not have been reconciled, because the disease of the cell lining and of the vessels, bone marrow and connective tissue spaces undoubtedly work together to permit the extension of the process to healthy cells, to the surface or to the endocranium. A mastoiditis, as we know it, is really an osteo-endosteal-periostitis with osteomyelitis fibrosa. During the course of the inflammation within the mastoid process, the inflammatory pressure is probably the greatest single factor, and by this term is meant not only ordinary mechanical pressure of the purulent exudate within the individual cell, but also biological pressure in its broader sense. In the early stage, the first pneumatic cell involved within the mastoid is filled with the exudate called forth physiologically, as a response to the inflammatory irritation. The organism, as a whole, then sends out the first troop of combative agents to meet the invader. There is an emigration of leukocytes and the exudation becomes purulent. The pressure within the cell is increased, the surrounding bone and connective tissue is involved, and there may be rupture into an adjacent cell made possible by destruction of intervening bone. The rupture into the adjacent cell relieves the pressure, but it also opens a new channel for further progress of infection, a new reservoir, as it were, and the entire process is repeated. The larger the common cavity thus formed, the less the possibility of resorption; and the edema and proliferative changes which ordinarily take place simultaneously lessen the chance of resorption still more. We find here, therefore, the explanation for the clinical observation that the extensively pneumatized mastoid process is more liable to marked involvement than that in which the pneumatization is restricted. There is also an anatomical reason for this, in that the spongy spaces separating the cells contain marrow and many vessels useful in resorption and retrotransudation; and it is evident that the larger and more extensive the cells, the smaller and thinner must be these dividing marrow spaces. As stated before, during the entire process osteoclasts and osteoblasts are at work and the destruction and reformation of bone goes hand in hand. Whether or not the presence of new bone predominates is an indication of whether or not the condition is reparable. The important clinical fact to be derived from this observation is that in the coalescent type of mastoiditis, the operation, if deemed necessary, must not be performed until a

definite line of demarcation by the new bone has appeared, and this takes place not earlier than the eighth day after the onset. If the mastoid is opened before this line of demarcation presents itself, there is a great possibility of future danger, severe complications, and the necessity for a second operation much more serious in character.

There is, however, an entirely different type of mastoiditis where early operation is not only permissible, but is definitely and seriously indicated. This is the hemorrhagic or thrombophlebitic form where the onset of the middle ear disease may be of the usual nature, and the paracentesis performed, but the general symptoms continue. There may or may not be the local sign of tenderness over the mastoid process and swelling of the soft tissue; and the less careful observer, led astray by the lack of local findings, may fail to make the very essential blood examination which is the informative factor in this type of invasion. This is the type of mastoiditis where the infection is centered in the minute blood vessels themselves, and where it progresses rapidly through the venules directly into the lateral sinus or through the dural plate, leading to severe and fatal complications within the first few days. This is the mastoiditis caused by the streptococcus hemolyticus; and, although this organism may be demonstrable in the bloodstream, it is not always so. But the positive blood culture is not necessary, for if the patient shows marked general symptoms, does not improve rapidly after the paracentesis and repeated examination of the blood shows a diminution of the red blood cells and a daily decrease in the hemoglobin, we, knowing what the streptococcus hemolyticus is capable of doing, and, realizing the serious nature of what is happening within the mastoid and to the individual, must intervene early and clean out the mastoid process thoroughly. An acute, hemorrhagic mastoiditis of this type with its mass of infected thrombi filling the venules of the mastoid process is comparable, in its effect on the individual, to a sinus thrombosis. It is in fact the same pathological condition in a disseminated sense, and just as no trained worker waits long after the diagnosis of sinus thrombosis has been established to correct the situation surgically, neither will an observer, cognizant of what is occurring, hesitate in such a case to intervene early and radically.

For a number of years the bacteriology of the mastoid was rather summarily dismissed from scientific consideration of the entire problem with the remark that "too little is known of this subject to permit of a definite statement." It is largely the contributions of Wirth, of the Heidelberg Clinic, which have aided us in this. In a statistical study, Wirth found that in 84 cases of acute otitis media, the causa-

tive organism in 53.8 per cent was the streptococcus hemolyticus, and in 23.8 per cent the streptococcus mucosus, the balance being caused by various types of pneumococci, streptococcus viridens, bacillus influenzae, staphylococcus and mixed infections, none of which seems to have particular virulence or to lead to severe complications. Wirth repeats what others of us have observed, namely, that the streptococcus mucosus is often quite difficult to diagnose. This organism, which in some clinics is so feared because of its insidious action and its tendency to lead to very severe complications late in the disease, and with comparatively few symptoms or signs, has no absolutely characteristic morphology. It may appear as a diplococcus or as a streptococcus, and apparently changes its morphology during its cultural growth. It is encapsulated, but the capsule, even by the so-called "specific stains" such as Thionin, is not demonstrable in every instance. Sometimes, when the capsule cannot be demonstrated by the Thionin stain, it becomes apparent when the Giemsa stain is employed. Good results have also been obtained by the Gram stain, using a longer after-staining of the smear with a dilute Fuchsin solution. The only criterion for this organism seems to be that it does produce a mucous growth on moist blood agar plate, but often only after the third replanting, and certainly only if fresh, *moist* blood agar is used. In Wirth's most recent work on the subject he declares that the streptococcus mucosus is identical with the Pneumococcus Type III. In this same work, as a result of the examination of 230 cases of middle ear suppuration, he finds the streptococcus hemolyticus culpable in 43.5 per cent, and the Pneumoccus Type III, or the streptococcus mucosus in 18.2 per cent, both of these figures being slightly lower than his first report. As to the respective virulence of these two major offenders, Wirth finds in one series, the streptococcus mucosus leading oftener to operation, and in the other series, the streptococcus hemolyticus. Throughout the literature there seems to be no agreement as to this point, and it remains necessary to consider the type of organism as only one factor, and to remember that the severity of its action depends on other factors, such as the anatomical condition, the location of the tympanic membrane perforation, the constitution of the individual, and the precurrent condition, the latter having especial importance since it has been found that, for example, an otitis caused by the streptococcus hemolyticus following a sore throat or a grippe produces complications oftener than one caused by the same organism following an ordinary rhinitis or a nasal operation. The otitis which is often seen following the placing of postnasal tampons, postoperatively, usually tends towards spontaneous healing.

There are many laboratory examinations which are not only useful but essential in the proper evaluation of a case of acute mastoiditis. To some of these, the years have added increasing value; some have decreased in importance. It would seem that the Roentgenographic examination has, in a good many instances, held an unjustifiably high position, because, in the final analysis, its chief value seems to be in determining the state of pneumatization, and only by repeated serial plates can one hope to gain much information as to the actual degree of destruction which the interior of the mastoid has undergone.

The lumbar puncture can be very informative. Neumann teaches that in order to aid in the recognition of an early meningitis, every acute otitis in which the fever persists after the paracentesis, should have a lumbar puncture with a careful examination of the fluid withdrawn. The significance of the cell count, and of the pressure determination, particularly following jugular vein compression in suspected sinus thrombosis, is obvious. It is well to remember, in addition to this, that a finding of glucose reduction in the spinal fluid is often the earliest sign of suppurative meningitis, being demonstrable before the appearance of the bacteria.

The examination of the blood sugar is important when we remember that with an increase of sugar in the blood, there is often a tendency to the production of an acute coalescent mastoiditis which may be painless and may cause a very slight temperature elevation.

The blood culture in a suspected case of sinus thrombosis is not reliable, particularly if it is negative, because it may still be in the early stages that the central portion of the thrombus is infected, with the outer layer sterile; and it must be construed as poor surgery to await the presence of a positive blood culture before operation in a case presenting other convincing signs.

Of all the laboratory examinations, the most important is the routine and complete serial hemogram before and after operation. The importance of the red blood cell count and hemoglobin determination early in the disease has been mentioned in connection with the acute hemorrhagic or thrombophletitic mastoiditis. Fortunately, this type of mastoiditis, however, is rare and therefore the part of the blood examination which offers us a more common help is the leukocytic determination, not, however, in the old sense of contenting ourselves with a simple white cell and neutrophile count, but—far better—by the use of the von Schilling method.

It may be well at this point to study the value of the von Schilling blood determination in general disease processes. I do not propose to

enter here into a discussion of the development of the various blood elements or into a consideration of the claims of the monolistic, dualistic or triolistic schools. Suffice it, for the present, to make the general statement that when the human organism is invaded by an irritating agent, there is called forth from the bloodstream the defensive force in the form of leukocytes. If the invader is of a mild nature, the defensive forces constantly at hand coursing through the blood stream are sufficient to overcome it. If, however, the invader is more severe, reserve forces are called for, and the hematopoietic

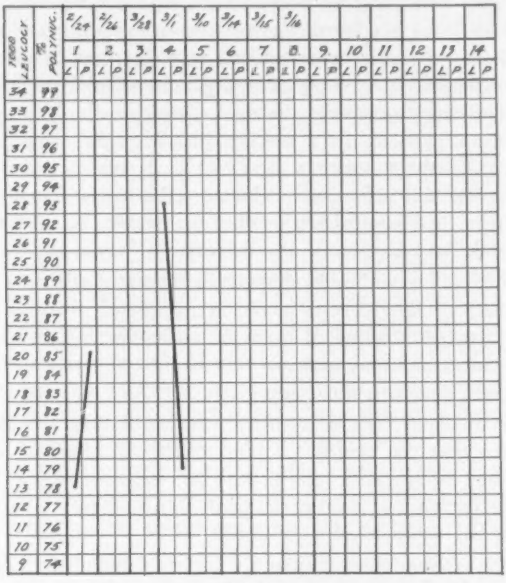

Plate 1.

apparatus sends into the blood stream an increased number of leukocytes. If the invader proves to be still stronger, then the demands on the defensive forces are such that the blood supplying apparatus must send out troops of youthful defenders before they have had time to mature, and in such a situation, the so-called "youthful" forms are found in the blood stream.

It was long since stated that the total white count was evidence of the resistive power of the organism and that the neutrophile count was an indication of the severity of the infection. McKernon has drawn up a chart showing what percentage of neutrophiles can

be expected normally, at every degree of leukocytosis. If, as shown by the accompanying example, the percentage of neutrophiles is higher than the "normal" limit at any stage of leukocytosis, it is a serious sign; whereas, if the reverse is true, *i. e.*, the line is in the "right" direction, it is indicative of adequate resistance on the part of the body (Plate 1). This chart alone, however, is not sufficient and, on the basis of Arneth's work, von Schilling has given us his convenient and significant hemogram and his conception of the "shifting to the left." This means that, whereas normally the neutrophiles are made up of 63 per cent segmented forms and 4 per cent of the so-called "staff" cells, or unsegmented, earlier forms; when an infection is present, the staff cells are increased. In a mild infection the percentage of staff cells rises to 5, 6 or 7; in a moderate infection to 10-15; and in a severe infection to 20, or even 30. This is known as the neutrophile or combat phase. If, during the course of infection, daily examination of the blood shows that the staff cell

Plate 2. Showing from left to right, myelocyte, metamyelocyte or youthful form, staff cell and segmented neutrophile. (Taken from Schleip-Adler's Atlas der Blut Krankheiten.

percentage remains high or increases, it is an unfavorable prognostic sign, and the appearance of these staff cells and of metamyelocytes or "youthful" cells constitutes a "shifting to the left," this term referring to the position in a tabular sense on the hemogram. As the infection is met and overcome by the resistance of the host, the monocytes increase in number and we have what is known as the monocytic or conquest phase. Following this, if reparation continues, the lymphocytes gradually increase and we have the lymphocytic or healing phase, which precedes the return to the normal blood picture and the recovery phase. It must be emphasized that a high staff count does not mean a fatal outcome, but a continued high staff cell count or a steady increase in the same does (Plate 2).

I believe that if these facts pertaining to general infection in the body are fully grasped and their significance understood, it will be unnecessary to comment further on the importance of such examinations in mastoiditis. Although this type of examination has not yet

attained to widespread use, many of us who have used it in our practice have seen its value, and have come to realize that it can offer us a clue, often long before definite symptoms otherwise have appeared. Not the least important is its value in determining the case which does not require operation, and of the charts which we are presenting herewith from our own observation, the last is particularly significant in this respect (Plates 3, 4, 5 and 6). In this case (H. D.), on March 22 the patient, although the discharge from the ear was decreasing, complained of unilateral headache and a throbbing in the affected ear. But, although the total white count at this time, the patient being an adult, was 13,500, in view of the fact that the staff cells, which had never been higher than 4 per cent, dropped to 0 with an increase in the lymphocytes, operative interference was decided against; and the subsequent improvement in the blood picture and complete recovery on the part of the patient again proved the worth of this type of examination.

Aside from these examples, I wish to quote briefly from the work of Dr. Else Levy, of the Charité Clinic in Berlin, who made a very extensive and thorough study of the von Schilling count in otogenous diseases. Dr. Levy emphasized that, although the blood picture evaluation is only one symptom, and that operative indication cannot be established on the basis of this examination alone, it is nevertheless a vitally important one, a valuable one and an informative one. She points out that of cases of mastoiditis with severe complications, those with brain abscess produce less influence on the blood than do sinus thrombosis, sepsis and erysipelas, and these less than those with meningitis. An important point practically is that in those very disturbing cases of pseudomastoiditis, where we have a swelling over the mastoid process sometimes to such an extent that the auricle protrudes, a discharge in the external auditory meatus, and a protrusion of the canal wall to such an extent that the tympanic membrane cannot be completely visualized, with an eczema or external otitis present—in other words, where we must attempt to differentiate between a genuine mastoiditis and extensive signs and symptoms from an external otitis, the von Schilling count can be particularly helpful, because it has been proven that an external otitis, as such, has no particular influence on the blood picture made by the von Schilling method, whereas a genuine mastoiditis, of course, has.

As yet, no mention has been made of the actual symptoms leading to a diagnosis of acute mastoiditis and of the necessity for operation. The typical case with continuing discharge (especially when pulsating); persistent fever (in children after the fourteenth day, and in adults after the tenth day); pain over the mastoid (that of less sig-

nificance in the first few days of the disease not to be confused with the indicative pain appearing later); thickening of the overlying soft tissues; sinking of the posterior-superior canal wall; the mastoid drum membrane (with the epitympanal perforation more dangerous than the mesotympanal); and especially with an outspoken subperiosteal, Bezold or Mouret abscess, or facial or abducens paralysis, needs, I believe, no particular comment. When, however, in the third week in children, or earlier in adults, most, if not all, of these symptoms have disappeared, but where the return of the hearing to normal is not so rapid or complete as we would desire, where the patient complains of throbbing or pounding, particularly if it is concurrent with the pulse, and of unilateral headache, especially when it is worse at night, we must be on our guard; and it is in these cases, the borderline cases, that the von Schilling count can be of inestimable value. With a sudden cessation of the unilateral headache, if the blood picture does not show a rapid return to normal, we must keep in mind the possibility of a pacchymeningitis externa.

Whether or not operation is to be performed "early" or "late" is a question that has always occupied a prominent part in discussion of acute mastoiditis. I offer you herewith a chart from a paper of Professor Neumann, of Vienna, which I present without further comment, inasmuch as it is doubly significant, coming as it does from the clinic which has, perhaps, the greatest otological material in the world today.

TABLE I—1302 OPERATED CASES.

	OPERATED IN WEEK									
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*Later than the eighth*	*Number Total*
Number of Operated Cases	28	185	245	254	151	110	56	74	119	= 1222
No. of Complications	16	62	82	99	67	57	30	38	68	= 519
Percentage	57.0	33.5	33.5	39.0	44.0	51.5	53.5	51.0	57.0	= 42.5
Fatal Outcome	7	12	12	11	5	3	1	8	10	= 69
Percentage	25.0	6.5	4.9	4.3	3.3	2.7	1.8	10.8	8.0	

TABLE II—422 OPERATED CASES—MAY.

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*Later*	*Total*
Number of Operated Cases	40	80	102	89	39	38	15	12	33	= 422
No. of Complications	8	30	32	35	23	15	8	4	17	= 172
Percentage	20.0	37.5	31.0	39.0	59.0	39.5	53.5	33.0	51.5	= 40.5
Fatal Outcome	4	8	6	5	2	1	—	2	1	
Percentage	10.0	10.0	6.0	5.5	5.0	2.5	—	16.5	3.0	

PLATE 3—CASE No. 5336 (L. A.)

Date	*W.B.C.*	*B.*	*E.*	*M.*	*Mt.*	*St.*	*S.*	*Lym.*	*Mo.*
Normal	6-8,000	1	3	—	--	4	63	23	6
5-15-29	15,450	—	—	—	—	12	69	15	4
5-16-29	12,700	—	5	—	—	8	66	18	3
5-17-29	9,900	—	4	—	—	6	64	19	7
5-18-29	11,650	1	4	—	—	8	55	28	4
5-20-29	8,300	—	5	—	—	10	48	28	8
5-22-29	8,750	—	3	—	—	11	39	35	12

May 23, 1929: Right mastoidectomy. Extensive coalescence, large amount of pus present, marked development of zygomatic cells.

PLATE 4—CASE No. 11880 (B. M.)

Date	*W.B.C.*	*St.*	*S.*	*Lym.*	*T.*	E.	*B.*	*M.*	*Mt.*	*C.I.*
Normal	6-8,000	4	63	23	6	3	1	—	—	—
4-15-29	22,100	9	63	22	5	1	—	—	—	.78
4-16-29	24,000	9.6	74.7	8	6.6	.3	—	—	.6	—
4-17-29	17,500	7	65	22	6	—	—	—	—	—
4-18-29	23,500	16	70	9	5	—	—	—	—	—
5-3-29	29,150	15	70	9	3	2	1	—	—	—

April 19, 1929: Left mastoidectomy. Pus in the antrum and periantral cells. Remainder of mastoid sclerotic.

May 3, 1929: Right mastoidectomy, entire postantral region and posterior tip cells filled with pus, with breaking down of cell walls in this region. Balance of mastoid filled with small cells containing serous exudate. Cortex thickened.

PLATE 5—CASE No. 10358 (B. C.).

Date	*W.B.C.*	*B.*	*E.*	*M.*	*Mt.*	*St.*	*S.*	*Lym.*	*Mo.*
Normal	6-8,000	1	3	—	—	4	63	23	6
4-10-29	16,100	1	1	—	—	10	47	30	11
4-11-29	16,400	2	2	—	—	9	53	36	8
4-15-29	14,250	1	—	—	—	7	44	34	14
4-16-29	14,200	1	2	—	—	4	52	26	15
4-18-29	14,200	1	—	—	—	4	53	31	11

No operation.

PLATE 6—CASE (H. D.).

Date	*W.B.C.*	*B.*	*E.*	*M.*	*Mt.*	*St.*	*S.*	*Lym.*	*Mo.*
Normal	6-8,000	1	3	—	—	4	63	23	6
3-15-30	13,700	0.5	2	—	—	1	64	31	1.5
3-19-30	13,300	0	2	—	—	4	72	21	1
3-22-30	13,500	0	1	—	—	0	68	29	2
3-28-30	9,800	0	1	—	—	4	41	51	3
4-2-30	8,700	0	2	—	—	1	54	41	2

No operation.

As to whether or not the mastoid cells must be completely exenterated, or whether the relief of pressure by the simple antrotomy is sufficient, can be rather conclusively settled, I believe, by reporting that in the Neumann Clinic, where complete exenteration of the mastoid cells in their entirety is routinely followed, secondary operations are rarely known.

I have considered it unnecessary to comment in detail on the classical signs and symptoms of acute mastoiditis as enumerated above, and have attempted to emphasize the importance of a constant

remembrance and consideration of what may be taking place within the mastoid; to call attention to the more important danger periods; and especially to remind you of the value of the more refined laboratory aids in diagnosis. It has not been my intention to leave the impression that I consider any of these procedures sufficient singly as a basis for diagnosis of the degree of involvement, or for indication for operation. We must always return to the realization that a patient is being treated, and not a mastoid process; that we are not "specialists" but physicians who specialize; and that the combined picture of the patient's general condition plus the local findings and, not to be forgotten, plus every diagnostic aid at our disposal, is necessary for the proper evaluation of the disease at hand. To forget any of these factors is to be severely criticized, and to disregard the essential laboratory examinations, such as repeated serial blood counts —preoperative and postoperative (in order to foresee any possible complication)—on the basis of considering it too elaborate, or difficult, or time-occupying, when, in reality, it is quite simple, is to overlook a valuable aid.

I trust that the points brought forth in this paper may prove sufficiently stimulating to evoke a discussion which will be valuable to us all, and to promote a more careful and intelligent consideration on the part of all of us of the problem at hand.

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ANALYSIS OF 100 CONSECUTIVE NASAL SINUS CASES TREATED CONSERVATIVELY.*

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For the purpose of "taking stock" concerning results of distinctly conservative treatments applied to the average "mine run" cases of nasal sinus trouble, I made a detailed analysis of 100 consecutive private cases since January, 1926. Many of the cases in this series had been seen at other times for some reason or other; some of them had been under occasional treatment because of sinus trouble.

In attempting to evaluate the evidence furnished by these cases, many difficulties were encountered. To assemble with any degree of accuracy data respecting effects of treatment is quite different from assembling surgical data, for example, concerning operative recoveries, the time required for healing, or mortality percentages. Impressions as to improvement have been based upon the findings upon physical examination, gain in strength and weight, together with the patients' reports as to relief of symptoms. In addition to upper respiratory examinations, the majority of records include blood pressure, urinalysis and general physical observations; also allergic, metabolic, cardiographic and other findings in certain cases.

The cases of this series presented, as cardinal symptoms of sinus diseases: *1.* Recurrent attacks of nasal inflammatory symptoms; *2.* local pain; *3.* mucoid or purulent nasal discharge; *4.* hypertrophic, atrophic, fibrotic, polypoid or necrotic lesions of the mucosa; *5.* turbinal turgescence; *6.* gross architectural alterations of nasal structures; *7.* headache, frequently of morning type; *8.* aprosexia; *9.* disturbance of lower respiratory, gastrointestinal, glandular or nervous systems; *10.* general debility.

There are records of blood findings in 38 per cent of these cases; among other findings were syphilis, brucella abortus and plasmodium malariae; primary anemia was discovered in one case and agranulocytosis in one cse. These findings, together with the differential leukocyte counts, are not included in the tabulation because they are of no statistical value, in view of lack of any record of blood examination in 62 cases.

The majority (80 per cent) were seen in acute exacerbations of pre-existent sinus trouble. Sixty-three cases had been previously

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operated, from several months to several years before. Attempts to chart operative effects on the patient were finally abandoned, after considerable effort, as it became apparent that any such statistical evidence would be so questionable as to be of no value.

Of this series, 65 were in full possession of tonsils; 32 of these gave no evidence, in history or findings, of tonsillitis; 14 more had some remnants of tonsils, or regrown adenoid tissue in the tonsil regions, without apparent trouble therefrom.

Systolic readings below 100 are recorded as hypotension; systolic readings above 150 are recorded as hypertension; no records of blood pressures were found in the records of 16 cases below the age of 18 years; the tension percentages being figured on 64 cases over 18 years of age.

In 39 cases there was evidence of ("allergic") special tissue-sensitivity to foreign protein. This evidence included history of respiratory, skin, or both, reactions to certain foods, of family sensitivity and of individual sensitivity; in many cases allergic studies had been made, but because of wide variations in these test-methods, attempts to incorporate the details into the charts proved impractical.

In 18 cases no pus was found in the nose on examinations; cytologic examinations by Sewall's technique were not made, the diagnosis of irritative or inflammatory disease of the lateral nasal wall tissues being established by gross findings and history; most of these showed occasional watery or glairy discharge, frequently also irritation of the transitional and skin areas at the introitus, in addition to cardinal symptoms of sinus disease. No etiologic relation is inferred between sinus disease and thyroid changes noted, though definite relations seem to exist between thyroid changes and hypertension. Omitting the general systemic febrile symptoms associated with acute phases of infectious sinus inflammation, there was little significant indication of commensurate etiologic relation between general systemic debility and sinus disease; that is, many cases showed local evidence of extensive suppuration and disease with relatively less systemic debility, and vice versa.

Thirty-two cases showed associated gastrointestinal disturbance, some of which were probably gastrointestinal expressions of the same tissue-sensitivity which, in the upper respiratory areas, was manifested as sinus trouble.

Almost 20 per cent showed chronic middle ear pathology; 13 per cent were arthritic; 4 per cent had associated pyelitis, only one of whom was an adult.

Morning headache was complained of in 52 cases; this varied from slight pressure and general "wooden-headed feeling" for an hour

or two to increasing ache up to 10 or 11 o'clock, subsiding by from 1 p. m. to 5 or 6 p. m.; 90 complained of difficulty in concentrating attentions for varying periods after rising.

DIGEST OF FINDINGS		AGES 7 to 72 average 37 yrs.	
Acute 1	Acute exacerbation 80	Monolateral 4 chronic 19	Bilateral 96
Tonsillitis	37 cases	acute 10	chronic 27
Pharyngitis	acute 3	chronic lateral 78	follicular 86
Gingivitis	acute 3	chronic interstitial 38	
Tension	hyper 10	hypo 22	
Thyroid	palpable 22	hypertrophy 18	
Ear	canalitis 12	tympanitis acute 3	chronic 19
Eye	ant. uvea 9	conjunct. 30	nerve 1
Arthritis	acute exacerbation 6	chronic 13	
Headache	acute morning 52	chronic occasional 20	
Gastro-Intest.	32		
Pyelitis	4		
Sensitivity	individual 19	family 20	
Purulence	71	acute purulent 11	
Polyposis	pellucid 4	fibrotic 3	mixed 5
Hypertrophy	simple 16	polypoid 5	
Atrophy	slight 11	moderate 5	marked 7
Previous Operation	T-A 35	septal 14 sinus 72	multiple 58
Lower Respiratory	trach-bronch. 25	bronchiect. 9	asthma 11
Cranial Nerve	retrobulb. 1	VII 1	VIII 2
Mening.-Thromb.-Abscess		None	
Osteomyelitis		None	
Aprosexia	90		

RESULTS

	30 days	60 days
Disappearance of gross mucosal inflammation	68%	18%
" " sinus pain	78	10
" " mucopurulence	64	6
" " turbinal turgescence	80	6
" " crusting or ulceration	68	6
" " headache	38	28
" " aprosexia	58	31
Gain in weight 52 cases	28	10
Improved blood-pressure 39 cases	12	13
Increased physical strength	79	
General improvement	79	
Intracranial complications	None	
Osteomyelitis	None	

After many failures to find some satisfactory expression of findings and results in the form of charts, attempts to express them statistically had to be abandoned because in each chart there appeared misleading numbers and percentages. The following "digest of findings" contains in tabulated form the outstanding items encountered in this series.

The table entitled "Results" sets forth certain items of improvement; no definite connection could be established between "findings," either individually or in groups, and "results." Many attempts were made to devise certain correlations of results with findings, but each had to be discarded as it became apparent that nothing definite materialized. The relations between general systemic manifestations of pathologic processes and their local nasal manifestations are of particular importance; the relations between sinus inflammations and sensitivities (allergies) are of equal importance; concerning these relations statistical analysis of the data furnished by this series of cases was too vague to be charted. The inclination to attribute certain results to certain treatments leads to the fallacies notoriously associated with *post hoc-propter hoc* deductions. Above all, this attempt to analyze records emphasizes the lack of any measuring stick which may be used gauging results following treatment.

The tabulation of "results" is made up of information assembled from the individuals constituting this series of cases, and should be clearly understood as attempting to establish nothing further than a chronicle of changes observed. The improvements noted, regardless of the specific reasons therefore, have been highly satisfactory.

In 11 per cent the upper respiratory tract was apparently the site of *local pathology only,* whereas in 62 per cent there were acute exacerbations of chronic upper respiratory dysfunction which were clearly and significantly *local* manifestations of *generalized pathology,* evidenced by changes in white cell count, temperature and metabolism; also in associated tissue-reactions in other localities—lower respiratory, circulatory and gastrointestinal tracts, ocular and aural tissues, tendons, synovia, lymphatic tissues and certain gland structures.

It seems that many of the disturbances which are commonly termed "infectious inflammation of the nasal sinuses" are not limited to nasal sinus tissues, even though the patient's chief complaints are directed to the nasal areas. Etiologic factors of different natures are involved, and these different factors manifest their effects in different places and phases during the pathologic disturbance. The incidence of *acute nasal* symptoms is almost always brought on by shock, exposure, chill, trauma, fatigue, digestive disturbance or contact with noxious environment. Evidence of *systemic disturbances* succeed the onset of local nasal symptoms after relatively short intervals.

The nasal disturbance is of the nature of a reaction between nasal tissues and micro-organisms, while the succeeding, systemic disturbances are reactions between more remote body tissues and noxious

emanations from the nasal areas. The nasal tissues in 90 per cent of this series were unquestionably in contact with pathogenic microorganisms during the relatively symptomless periods between acute exacerbations. There is apparent a somewhat uniform sequence of pathogenetic episodes in a large number of cases. Acute sinus trouble is practically always precipitated by some *physical* disturbance of normal working conditions of the body. The local changes of a *biochemical* nature which follow are signalized by disturbance of hitherto quiescent relations between nasal tissues and their microorganismal residents. This is explainable only upon a basis of alterations *in the nasal tissues themselves* as it is inconceivable that chilling the ankles or breaking the femur will deposit new bacterial flora in the upper or lower respiratory areas to set up acute sinusitis or pneumonia. Wright[1] has said that the skin is no less the cause of dermatitis than the tubercle bacillus is the cause of tuberculosis. Without tubercle bacillus one does not have tuberculosis—without one's skin, well there is no use wasting breath on that. There seems to be good reason for believing with Piness[2] that some infections take place when opportunities are afforded by acute allergic nasal reactions, regardless of bacterial nature of the allergy. In these observations is contained much food for reflection upon the nature of the primary tissue-reactions which are noted in sinusitis. The products disseminated throughout the system from the nasal areas, once an acute local pathologic process is established, are more or less toxic and their general nature is well understood. One of the main problems in sinus infections seems to be the restoration of that previous condition of the tissues which was characterized by compatibility with allergens or bacteria. The general nature of the biochemical changes converting compatibility into incompatibility is that of acidosis; this has been fairly established by the numerous biochemical studies of tissue changes in acute inflammation. The problems of acidosis are legion and are entirely aside from the subject-matter to be presented in this paper. Disregarding all details concerned with the acidotic process itself, antiacidotic measures are clearly indicated, among other therapeutic attempts to meet the general situation. This indication has been the backbone of the conservative treatments which have been applied in this series of cases.

Under "conservative" treatment is included certain *surgical measures*, namely: resection, synechia removal, or reconstruction of septum; simple incision of antrum, bulla and sphenoid in giving vent to purulent content; removal of polyp, tonsillectomy and adenectomy.

Nonsurgical Measures:

1. Suitable regular exercise.

2. Dietary regime: *a.* Elimination of foods known to disagree with patient. *b.* Selection of foods representing good vitamin balance. *c.* Selection of foods representing proteid, fat, carbohydrate balance. *d.* Fluid intake approximating 8 ounces hourly per 10 pounds of body weight every day.

3. Periods of alkalization, as evidenced by neutrality or slight alkalinity of urine (alkalinity by no means precludes the possible existence of acidosis).

4. Periods of iodization, always controlled short of sharp focal reactions.

5. Use of salicylates sufficient to control pain.

6. Use of physiotherapy, by hot fomentations, radiotherapy or diathermy.

7. Local therapy, by tamponade (organic silver salts, or hygroscopic, or both), negative pressure alone or in conjunction with tampons, medicated steam inhalations, with or without oil nebula.

8. Mechanotherapy, interdiction of hard nose-blowing; habituation to cleaning nose by suction down nasopharynx; negative pressure.

9. Avoid all nose washes (Dobell's, Seiler's, salt, soda, etc.); avoid insufflations of water in swimming; avoid diving or submerging the head in sea, pool or tub bathing.

10. Continue regime for months, regardless of prompt subsidence of symptoms; resume regime at intervals, even in absence of symptoms.

In special cases, endocrine imbalances indicate thyroid, insulin, pituitary or other suitable endocrine therapy; marked anemias require suitable additions in the form of drugs or diet; asthenia requires regulation of sleep and rest periods; overwork, worry, emotional or psychic disturbances, and any coincidental condition disturbing the general health, such as rickets, Malta fever, malaria, tuberculosis or syphilis, must be controlled and eliminated as far as possible.

I wish to call special attention to the surprising value of regular, suitable exercise. I have had many patients declare their morning setting-up exercises, shower and rubdown do them more good than any local treatment. Of course, the details must be worked out for each individual respecting exercises and cold baths. Hot baths, foot baths, sweats and massage are also of definite value in many cases in conjunction with other measures.

In my experience a balanced diet, with adequate vitamin intake, especially A and D, is particularly needed by many patients. It is seldom sufficient to leave the selection of food to the patient. The patient must be made to understand that the importance of proper

diet does not extend over only a few days or weeks, but constitutes an essential factor in the maintenance of health and freedom from nasal sinus disease.

Alkalization may be usually accomplished by diet. Some cases, however, are more satisfactorily alkalized by some such means as citrate and bicarbonate of soda, Wright's powder or citrocarbonate, taken with fluids three or four times a day.

Fluid intake should be largely between meals, usually at the rate of a glass an hour. The taking of a large fluid intake in itself usually produces a decided antiacid effect.

Iodization must be approached cautiously; some cases can take only one dose at the beginning on account of lymphatic, skin and glandular focal reactions. Under supervision, iodid can be increased gradually without producing undue reaction until two or three doses per day are taken from three to five days at a time, producing a tolerable measure of rhinorrhea, with or without sense of activity in the parotid and cervical regions, perhaps also slight headache, abdominal discomfort or skin reactions. Most so-called "idiosyncrasies" subside under careful iodid administration.

Salicylates may be used with good advantage for control of pain and headache in connection with the treatment herein outlined. Aspirin in doses of 1 grain per 10 pounds of body weight, powdered up with 20 to 30 grains of bicarbonate of soda, followed by absolute rest, has proved satisfactory; this dose may be repeated if no amelioration of intense pain has been noted after 30 minutes. Morphin and opium should be avoided because of danger of addiction and of the undesirable antagonistic effects upon tissue reactionary processes. Atophan and phenacetin may be often combined advantageously with salicylate.

Physiotherapy in a form to stimulate rather than depress tissue-reactions — in other words, to supply energy rather than abstract energy—has decided value as adjuvant measures in conjunction with the systemic attack outlined here. Heat is the most readily availabie form of energy for this purpose. Local therapy in the form of tamponade, negative pressure and medicated steam often proves of decided value, especially at the beginning of a course of systemic treatment.

The patient should be trained to avoid blowing the nose hard, learning to clean away tenacious secretion from the upper respiratory tract by drawing it backwards into the nasopharynx, to be expectorated.

The habitual use of alkalin or other nose washes is to be discouraged, because of the undesirable effect of quick evaporation, which

puts a stop to ciliary activity. Occasionally a nasal douche may be necessary, but it is always succeeded by a period of dessication of the cilia, during which secretion lies unmoved, evaporating into films and crusts which again demand lavage for their removal. The same is true of water insufflated while swimming or bathing. Cocainization produces paralysis of ciliary activity, a particularly undesirable condition of the affected nasal areas.

Most important is to impress upon the patient at the start that this general regime is to be kept up, more or less as outlined, for months or years, regardless of improvement.

I have had opportunity to observe the condition of patients over periods of years under the influence of such a general regime as has been outlined; in comparison with similar cases treated by radical surgery or by local nasal treatments, such as antral lavage, cautery, tampons and topical applications, the results of the measures herein advocated are definitely superior. The incidence of serious complications, such as osteomyelitis, meningitis, brain abscess, thrombosis and septicemia, in my experience has fallen to *nil;* during the past 10 years I have not seen one case so treated develop intracranial complication. I have seen such complications in many cases during this time; they were, without exception, postoperative complications. While I am prepared to believe that such complications may arise without previous surgery, I have not seen one in 10 years. Regardless of what *might* happen in this respect, it is *certain* that they are much more apt to occur under the conditions usually obtaining in the average course of radical nasal surgery.

Summing up: Upper respiratory conditions alluded to in this paper are made one with the general systemic conditions under which they develop. Co-operations on the part of general systemic mechanisms of resistance and recovery are solicited to a maximal degree in coping with disease conditions. The essential upper respiratory tissues are to be given all possible opportunities to regain normal condition, and to conserve functionability to the utmost, not only for present use, but for withstanding similar vicissitudes of the future. The upper respiratory tissues constitute an important part of the general mechanism of immunization and, as such, their preservation has important bearing upon the general health of the individual.

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1154 Roosevelt Building.

ONE METHOD OF CLOSING AN ETHMOID FISTULA.

DR. H. M. CUNNINGHAM, Vancouver, B. C.

The method employed in this case may have been used and reported by others; if so, it has escaped my notice. Having served me so well, I should like to pass it on.

A Finnish girl, age 19 years, came to me with a large fistula in the ethmoid region on the left side, of six years' duration. She kept it plugged with cotton, partly for cosmetic reasons and partly to prevent an outpouring of pus. There was complete nasal obstruction on both sides, due to a fibroid tumor that completely filled her nasopharynx. There was a purulent discharge from her left nostril and mucopurulent from the right. The vomer had disappeared; the posterior portion of the septum being represented by a band of membrane which had originally covered the edge of the vomer. Middle turbinate on left side was gone. On transillumination both antra were dark. The left lower eyelid was everted and drawn down into fistula.

The patient was emaciated, anemic and septic. Two unsuccessful attempts had been made (in Finland) to close the fistula; one by taking a flap from the bridge of her nose, and the other by a flap taken from beneath the eye. It is obvious that a skin flap could not possibly live over a hole the size of one's finger, even had it not been pouring out pus. Apparently the tumor and the sinus disease had been overlooked or disregarded.

Unquestionably, the cure of such a complicated condition would be a long drawn out affair. The first step was the removal of the tumor. The absence of the vomer was a great advantage here, for after snipping away the string of membrane I had free access to the growth. This was removed piecemeal with the cold wire snare. The tumor was so dense that the knobbed nut on the handle shaft could not be turned without a spanner, which I had made for a previous case. Having provided free nasal respiration, the sinus infection was cleared up by doing a radical operation on the left antrum, removing the remaining ethmoid cells, and a Mickulitz operation on the right antrum. Fortunately, we succeeded in getting a clear nose and were in a position to attempt the closing of the fistula. The problem was to secure a solid foundation for a skin graft. The

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absence of the middle turbinate precluded the use of that. It occurred to me to use the tip of her little finger, to which she readily consented. The nail was removed and a slanting incision made through the skin, beginning distal to the matrix and including the ball of the finger. Both edges of this were undermined, making one cuff for the inside and another for outside. After freeing the lid margin by an incision extending to the outer third, and removing cicatricial tissue, the tip of the marginal strip was anchored at the inner canthus in proper position. The edge of the fistula was then thoroughly freshened, the tip of finger inserted and securely sutured in place. The arm and head were immobilized by a plaster cast. Eight days later, the finger tip was amputated, leaving a complete

occlusion of the fistula, and a solid bed for skin graft. When well organized and in proper condition, a Thiersch graft was successfully applied, bringing to an end a series of procedures that had extended over nine months.

The final result was entirely satisfactory to both the patient and myself. She regained health and happiness, and was married within a year.

I regret that I have but one illustration; taken just before the plastic work was undertaken. This does not picture the wretched condition of the patient when she came to me, for she had gained 20 pounds in weight and could breathe with her mouth closed. The work was done 20 years ago, and unfortunately the photo showing the final result cannot be found.

614 Birks Building.

PRESENT STATUS OF THE LOTHROP-SKILLERN RADICAL OPERATION ON THE FRONTAL SINUS, WITH SPECIAL REFERENCE TO TECHNIQUE.*

DR. HARRY B. GOODSPEED, Philadelphia.

In presenting this paper, I lay no claim to any of the modern changes made in the radical Lothrop operation. I only represent the class in otolaryngology of the Graduate School of Medicine of the University of Pennsylvania, who have been taught this newer operation in the classroom, practiced it on the cadaver in the dissecting room, and observed it and assisted at it many times in the clinic. And, in so representing my class, I hope to reflect to you, as a mirror, our teachings during this past school year.

The original Lothrop operation, which appeared in 1914, was similar to, but an improvement over, the older Hallé operation, which had been recommended seven years previously. In this newer operation you will remember the intranasal operation of removing the middle turbinate and cleaning out the anterior ethmoidal cells was performed eight to 10 days prior to the more radical operation. At the second stage an incision was made in the eyebrow, the frontal sinus opened at the naso-orbital angle, the sinus cleared only of inflammatory tissue, the nasofrontal duct located and enlarged anteriorly with a burr or drill, a large opening made in the interfrontal septum, and a portion of the internasal septum removed just below the sinuses to a depth of 1 inch. The wound was dressed with a pressure bandage to aid the falling-in of the soft parts.

I do not intend to go into this operation in detail, but to mention only these salient points, so that you can readily see the similarities and changes in the newer operation.

The Killian operation you are, of course, all familiar with.

Before taking up the technique and steps of the Lothrop-Skillern radical operation, let me sum up what we believe to be its advantages over other radical frontal operations.

1. It is by far a simpler and less dangerous operation than the Killian, requiring one-half the time, less surgery, less anesthesia, less bone resection, and thus causing less shock to the patient. There have been no fatalities in over 150 cases in this clinic. In the Killian the mortality is stated to be 2 to 3 per cent.

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2. The eye is not disturbed except for the cutting of certain blood channels to the upper lid, which causes only a transient swelling and edema. There is thus no danger of diplopia, as is always to be thought of in the Killian.

3. Blindness has been reported only in those cases in which the sphenoid has been curetted at the same time, and we believe that injury to the optic nerve took place in these cases through a thin sphenoidal wall.

4. As the lacrimal bone and membrana papyracia are not disturbed, there is no danger of injury to the lacrimal duct or of epiphora.

5. The use of a burr to enlarge the nasofrontal duct has been replaced by the rasp and chisel, which seems by far a safer procedure.

6. Closure of the nasofrontal duct by falling-in of the soft parts, and the return of symptoms of blockage cannot occur as the bony wall, and thus the support, is not removed.

Likewise, there is good opportunity for granulations to close in the duct to its normal size by the time regeneration of the lining mucous membrane of the sinus has taken place, and thus we have as near a return to normal after the operation as we know how to obtain.

7. Hemicranial anesthesia, or neuralgia, due to cutting of the supraorbital nerve, is the same as in any of the other operations. However, no such cases have been reported in our clinic. All cases of hemianesthesia, which is so often seen immediately after operation, have cleared up after a few months.

8. The operation is far less disfiguring than the Killian, and also of a distinct improvement over the original Lothrop operation, as specific steps are taken to prevent the falling-in of the soft parts.

9. The danger of fistula formation is reduced to a minimum as there is no external drainage.

10. The continuation of symptoms due to the neglect to remove all diseased tissue or overlooking supraorbital ethmoidal cells is the same as in any operation.

11. The patient is more willing to undergo a radical operation when it is known that the eye is not to be disturbed and that usually he can expect to leave the hospital in eight to 12 days.

Before recommending this operation, the patient is always given the opportunity to obtain relief from his symptoms by the intranasal removal of the anterior end of the middle turbinate and curettement of the anterior ethmoidal cells. After this aid has failed, and in the emergency cases, the operation under discussion is the procedure of

choice. In emergency cases, the intranasal operation must be performed before going ahead with the external operation.

Unless the patient has been hospitalized, he is permitted to sleep at home the night before operation. He is instructed to take a laxative before retiring and to eat a light breakfast of fruit and cereal, but no meat, before coming to the hospital.

Upon his arrival at the hospital, a final checkup is made. The Roentgenographic study has, of course, been made before this time. If the eyebrows are heavy, they are both clipped short, but not shaved. Two hours before operation, the patient receives 10 gr. of sodium barbital to allay restlessness, the effect of morphin being saved for postoperative care.

On the operating table, the field is prepared with soap, water, iodin and alcohol. The head should be supported with sand bags, and it is essential to have good light, which can be reflected down into the sinus so that every corner can be examined during the operation. Ether by inhalation and the usual machine to deliver it by a tube to the mouth, in combination with a suction apparatus, is used during the operation.

Slides: 1. The initial incision is started at a point just below the inner end of the eyebrow, over the suture line of the ascending process of the superior maxillary bone and the frontal, and extends outward in the center or lower part of the eyebrow, for nearly its entire length. The Roentgenographic plates are in a frame, where the operator can refer to them from time to time, and the size will govern the length of the incision.

This first cut extends only through the skin and subcutaneous tissue, and these parts are dissected upward as a flap, for about ½-inch, and downward to the bony supraorbital ridge.

Any hemorrhage from the frontal, supraorbital or angular branch of the lateralis nasi arteries requires ligation with No. 0 plain catgut. Other oozing points are controlled with hemostats.

2. Beginning now at about ¼-inch higher level, the incision is carried down through the periosteum to the bone, and this flap is dissected and retracted upward and inward to the estimated extent of the sinuses. The lower flap is dissected downward only about ¼-inch so that a little bone may be removed here without injury to the periosteum, and a good, healthy edge will exist for suturing.

The reason for making these two flaps in this manner will be evident when we close our wound.

3. While the assistant holds the flaps with four prong retractors, the operator uses a small, half-round chisel and an opening is made

above the supraorbital ridge immediately above the frontal articulation with the superior maxillary bone. By cutting directly inward, a small button of bone is formed which can be pried out and a small hole is obtained for exploration of the sinus with a probe.

4. This exploration should be made carefully upward and outward, and the measured extremities of the sinus indicated on the exposed bone. This gives the limits for the removal of the anterior wall and if the chiseling is begun at these points, the sinus can be uncovered quickly and safely in very short time.

5. An angular Killian V-chisel is now used to make a groove, beginning at the outer measured limit of the sinus and on a line with the periosteum incision. Care must be taken not to injure the lower ¼-inch flap of periosteum during this procedure. The inner end of the groove will end at the hole already made. The V-chisel is used only until there is a slight crack running the entire length of the groove. Now, flat or large, round chisels are used only on the upper bone flap until this crack is enlarged sufficiently to permit the use of rongeurs or suitable bone-biting instruments.

6. The entire anterior wall of the sinus is thus removed, from its outer and upper confines to the intersinus septum, care being taken not to injure the supraorbital ridge.

The diseased portion of the sinus mucosa is now removed with curettes, and the underlying bone minutely inspected for any signs of necrosis. All ramifications are opened, and all partial septae removed, thus making the sinus one large, smooth cavity.

The Roentgenographic plates are referred to and studied in conjunction with the opened sinus. Any abnormalities shown by the plates are explored and, if found, opened in their entirety, thus throwing them into the sinus cavity.

Particularly careful study is made at this time for supraorbital ethmoidal cells, infundibular cells, a frontal bulla, cells in the agger nasi and septal cells. If any of these cells be diseased, as they so often are in frontal sinusitis, and are overlooked at the time of operation, the results will in all probability be a failure.

7. A probe is now passed down the nasofrontal duct, and alongside of this a small rasp is guided into the anterior nares. The probe is removed and the ostium is enlarged forward and toward the septum.

8. As soon as more room is gained, a larger rasp is employed, or a bayonet chisel, to remove the nasofrontal spine. When rasping, care must be used not to jam the back of the rasp against the posterior wall, as the cribriform plate can be very easily fractured in this way.

At this stage of the operation, either the posterior nares should be packed or an assistant should carefully keep the nasopharynx clean from blood and fragments by using the suction tube through the mouth.

After the passage has been sufficiently enlarged to admit a piece of rubber tubing the size of a No. 26 Fr. catheter, all rough edges are smoothed off, and then a strip of gauze is threaded through and pullled back and forth several times to collect in its meshes all fragments of bone and loose pieces of mucous membrane.

The duct should be enlarged sufficiently to permit free drainage during healing. After this, we rather hope it will become smaller, so as to be approximately normal in size again. The diagram here indicates too large an opening. This may appear ideal but, in all probability, such a large opening would keep the sinus continually infected and it would drain forever.

10. The bony edges of the sinus are now carefully smoothed and bevelled off, so if there is any sinking-in of the skin, there will be no sharp edges sticking upward, causing unnecessary disfigurement. Some bone is removed from the lower edge during this step, which gives more periosteum for suturing. Care must be exercised here not to remove too much bone as the supraorbital notch may be deep and thus render this ridge weak and liable to fracture, either during the operation or from a trivial blow after the patient leaves the hospital.

11. A piece of rubber drainage tube is now passed down through the ostium into the nose. The upper end, being bevelled, is placed at about the center of the sinus. Sometimes a stay suture is brought from the upper end up through the upper skin flap and tied loosely. However, if the tube fits snugly and is trimmed off close with the nares this is unnecessary.

12. The upper and lower flaps of periosteum and attached muscle are now brought together and sutured with 10-day No. 1 catgut. This, you see, forms a strong support over the sinus, and its purpose is to prevent sinking-in of the soft parts, with the resultant ugly deformity.

13. The skin flaps are now brought together and sutured with fine silkworm gut.

If you will recall the manner in which the original incisions were made, you will see that this brings the skin suture line slightly lower than the periosteum and muscle flaps suture line. This is to minimize the possibility of the occurrence of a fistula.

After cleaning the operative field free from blood, a strip of iodoform gauze is placed over the incision. Over this is placed several layers of vaselin gauze, and over all is securely fastened a light, dry, fluffy dressing.

One or two hypodermics of morphin may be required during the night. The next day, the dressings are generally found saturated with blood and serum, and should be changed, and usually again in two more days.

No intranasal care is required.

Ordinarily, with a normal temperature, the patient is allowed to sit up on the fourth or fifth day, and the tube is gradually worked down until it is out by the seventh or eighth day, when the patient is allowed to go home.

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4111 Walnut Street.

A SATISFACTORY WATER SUCTION.

DR. PIERRE VIOLE, Los Angeles.

The apparatus as described below was devised in order to have negative pressure in my office without the disadvantages of the existing methods.

The objections that I allude to are as follows: In the water suction apparatus available up to the present time the water flows into the bowl or sink. This to my mind is objectionable because it is untidy, on account of the water splashing, which either wets or soils its surroundings. The noise produced is a disturbing element.

In consequence of the above objections, I consulted J. J. Cantor. I suggested the construction of a water suction apparatus with the

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exit into the trap below the ordinary bowl or sink. After considerable experimentation, we devised the apparatus as illustrated. To our great surprise, we have been able to obtain 28 inches negative pressure, with the local city water supply. As a gauge is attached, one knows exactly the amount of suction one is obtaining and this is conveniently regulated by either turning the water off or on. It is absolutely reliable, in that it has no moving parts which need oiling or care. It takes up no office space which can otherwise be utilized.

The volume of vacuum is certain, in that the possibilities of leakage from mechanical joints, the slipping of belts or the tubes being clogged by secretions or blood, are eliminated. The initial cost is small compared to the other contrivances available and can be installed easily and cheaply by any plumber.

As there are undoubtedly others who have been in search of this type of apparatus, I wish to pass the word along and do not hesitate to most highly recommend it.

304 Wilshire Medical Building.

CARCINOMA AND TUBERCULOSIS OF THE LARYNX. CASE REPORTS AND AUTOPSY FINDINGS.*†

Dr. Charles K. Shofstall, Kansas City, Mo.

Two cases are herewith presented, the first a squamous-cell type of carcinoma in the larynx and pharynx, and the second a generalized miliary tuberculosis with extensive involvement of the larynx.

Case 1: W. D., male, age 66 years, white, occupation unknown, was admitted to St. Margaret's Hospital in December, 1927, complaining of pain in the throat and on the left side of the head. Other complaints were pain in the right thigh and difficulty in moving the right leg. He had been having pain in the throat for two months prior to entrance to the hospital; he had noticed hoarseness some time previous to this but could not say for how long. The pain became so severe that he was unable to swallow liquids without strain or to speak without discomfort. During this time he had frequent sharp pain in the left ear, which was throbbing in character. It became necessary a few weeks before his death to feed him through a gastrostomy wound, due to inability to swallow, caused by the attendant pain and spasm produced by deglutition. He died three weeks after entrance to the hospital. The Wassermann and Kahn tests were four plus; other laboratory findings were of no particular interest. We were unable to obtain any record of treatment or examination afforded the patient previous to coming to the hospital.

Autopsy: Body is that of a fairly well nourished male, age about 65 years and weighing about 160 pounds. The abdomen showed nothing unusual. In the chest the lungs were markedly emphysematous (due probably to the choking and straining); in the upper lobes there were small masses of healed tubercles. There was hyperplasia of the villus papilli and sublingual tonsils. In the pharynx was seen a mass of tissue with considerable surface ulceration; the mass involved the surface and cartilage of the epiglottis and extends to the glottis along the anterior surface of the larynx; the tissue mass is seen to involve the left arytenoid and the false cord on that side; the left vocal cord is replaced by a hard, rubbery mass of tissue and there is partial invasion of the right true cord. Over the anterior

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†Autopsy findings are those made during service in the Department of Pathology at the University of Kansas.

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portion of the larynx the mass measures about 4 c.m. in diameter and is seen to extend well into the surrounding structures of the neck; this fungating mass invades the mucous membrane and pharyngeal muscles about the esophagus. On microscopic examination the section shows a very unusual squamous epithelial growth. There are seen four or five layers of undifferentiated squamous epithelial cells placed over irregular papillary-like processes. Considerable surface desquamation and necrosis is noted. Dense infiltration of dark, undifferentiated cells may be seen extending into the underlying tissue. There is no tendency to pearl formation. Section through the epiglottis shows massive infiltration of the cell mass into the cartilage

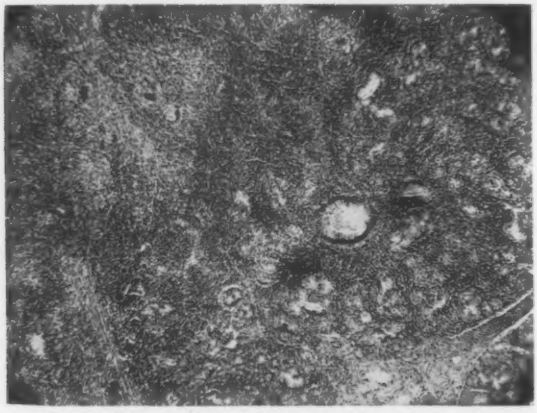

Fig. 1. High-power (X76) of section taken from the anterior aspect of the neck in the region of the thyroid gland. Numerous giant cells surrounded by epitheloid cells and a peripheral zone of round cells are seen. In some areas the tuberculous mass invades the thyroid gland.

and the same in the region of the thyroid cartilage. There is a considerable amount of secondary inflammatory reaction on the surface and scattered throughout the various sections of the mass. A few areas show perivascular round-cell infiltration.

Nothing else of particular interest is to be found in the autopsy aside from the parenchymatous degeneration of the heart, liver and kidneys, and atherosclerosis of the aorta. The kidneys were considerably affected and showed on microscopic section a marked swelling and edema of the tubules and the epithelial elements.

The record of the second case is as follows: *History:* G. M., age 55 years, white, male; occupation, matttress worker; was first seen in the clinic in March, 1925. At that time he complained of attacks

of coughing, pain and tightness in the chest and difficulty in breathing. He had typhoid 15 years ago and history of syphilis contracted 24 years ago. Examination of the patient's ear, nose, throat and larynx at this time was negative. A Wassermann taken at this time was four plus. He was next seen a year later—March 18, 1926. He came in complaining of hoarseness at that time. Examination then showed the larynx to be edematous, with inflammation of the arytenoids and false cords. The true cords were normal. He was next seen seven months later—Oct. 21—and at that time there was a small ulceration on the posterior third of the left vocal cord. The cords were adherent in the anterior two-thirds. He was not seen again in

Fig. 2. Low-power (X17.4) view of the carcinomatous mass in the neck. The tissue is extremely cellular, hard and rubbery. The growth is seen to invade the surrounding tissue by finger-like projections.

the clinic for more than a year—Nov. 17, 1927—and at this time examination showed a small ulcer on the left false cord. From then on, the ulceration developed until it involved both false cords, and finally before he died it was necessary to perform a tracheotomy on account of the marked obstruction to breathing through the larynx. During the last six months of his attendance at the clinic he had treatment with salvarsan and bismuth, about 36 injections in all. The Wassermann and Kahn reactions remained, however, persistently four plus. For a period the larynx improved somewhat under this treatment and the patient was able to breathe with less difficulty. Later he received intralaryngeal instillations of 20 per cent chaulmugra oil and some improvement was noted with this treatment. Dur-

ing the course of his treatment and observation the diagnosis was syphilis and tuberculosis of the larynx. Chest examination while in the hospital was never satisfactory, due to the noisy breathing. Laboratory examination showed three plus albumin in the urine, with some blood and pus cells. The patient died on Feb. 10, 1928, almost three years after his first visit to the clinic.

Autopsy: Body is that of a man about 6 feet all, weighing about 165 pounds, of good nutrition and normal muscular and skeletal development. There is a low tracheotomy incision in the neck, with the opening into the trachea patent. Old healed scars are seen on the glans penis. Removing the lungs shows them to be filled with

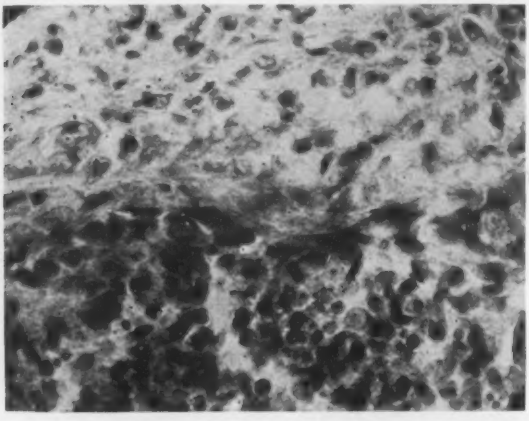

Fig. 3. Very high-power (X920) view of the cellular structure of the cancer mass. The cells are embryonal in type and vary in size, shape and staining reaction. There are a few mitotic figures and a considerable amount of round cell infiltration throughout the section.

miliary tubercles; numerous adhesions to the chest wall on both sides are noted. The larynx shows absence of the true and false cords, with replacement by inflammatory tissue and healed scars; cut section through the tissue reveals yellowish, rather caseous tissue, in the form of tubercles. The inflammatory reaction is rather widespread in and about the larynx and involves even the thyroid in a mass of inflammatory and caseous tissue. Miliary and conglomerate tubercles are found in the prostate, adrenals and kidneys; the small and large intestines show numerous tuberculous ulcerations of varying extent. The aorta shows longitudinal wrinkling with atheromatous plaques in all portions; no dilitation or aneurysm is found.

Histological Pathology: Examination of the tissue from the larynx shows the stratified epithelial layer to be proliferated and replaced in places with chronic granulation tissue. Beneath this is seen numerous tubulo-alveolar glands, which are replaced in many places by miliary tubercles. Numerous giant cells are seen and fibrous tissue is prominent about the affected areas. The vessels are thickened and prominent. The secretory glands are separated into lobules by thick bands of fibrous tissue. Some perivascular round-cell infiltration is noted here and there but it would be impossible to make a diagnosis of syphilis from the tissue, although it cannot either be excluded, and one may infer that it played an accessory role here and possibly a prominent one in the etiology of the lesion in the larynx. Complete anatomical diagnosis may be summed up as follows: Tuberculous ulceration of the larynx with absence of vocal cords; miliary and conglomerate tubercles of the lungs, prostate adrenals and kidneys; tuberculous ulceration of the large and small intestines; chronic hepatitis with fatty degeneration; chronic adhesive pleurisy; syphilitic mesaortitis; diverticulum of the bladder.

Discussion: In the first case the history and findings previous to postmortem examination are rather meager, yet one would be inclined to class this case as one of extrinsic carcinoma of the larynx, which, according to McKenty, St. Clair Thomson, Milligan and others, has a much more rapid course than the intrinsic type and is 100 per cent fatal. Much depends upon early diagnosis of the lesion. However, the extrinsic type may give early, only minor symptoms, or none, and this fact, together with extension rapidly into important structures in the neck, account for the high mortality. Pain is a late symptom in malignant lesions of the larynx. When this patient presented himself to the hospital for the first time, pain was the chief complaint.

The second case presents many interesting features, chief among which is the presence of a combined syphilis and tuberculosis of the larynx. It would be impossible to exclude syphilis here as a contributing factor to the later extensive tuberculous involvement of the larynx. It can probably safely be said that with the presence of a generalized miliary tuberculosis and the histological pathology findings at autopsy, that the tuberculosis of the larynx was the essential lesion.

Both cases had repeated four plus Wassermann and Kahn tests. This fact added to the difficulties in making differential diagnosis in either case, but more so in the second case, where both tuberculosis and syphilis were diagnosed and the patient treated accordingly.

St. Clair Thomson wrote many years ago concerning the necessity of the exclusion of syphilis in cases of carcinoma of the larynx, and other writers have mentioned briefly the possible relation of syphilis as an etiological factor in the formation of carcinoma of the larynx.

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3820 Genesee Street.

SOME EXPLANATORY REMARKS REGARDING THE STATEMENTS MADE PERTAINING TO THE DISCUSSION OF IODIN POWDER (SULZBERGER).

Dr. M. D. Lederman, New York.

In a recent discussion before the Otological Section, New York Academy of Medicine, October, 1929, in connection with iodin powder (Sulzberger) as used by the writer for 15 years in the non-surgical treatment of chronic suppurative otitis, the following statement was made (see The Laryngoscope, p. 311, April, 1930), "that the original iodin dusting powder patented by Dr. Sulzberger was one of a number of similar preparations, and that ulsanin, a German product of the same composition, had been used for several decades." I had never heard of such or similar preparation, neither have any of my colleagues who have become familiar with iodin powder (Sulzberger). The German product, ulsanin, referred to in the discussion of my paper is entirely different and described as hydrojodoborat, evidently a chemical combination containing iodin, while Dr. Sulzberger's powder contains finely impregnated boric acid with free iodin deposited from a solution of iodin, not in chemical combination.

Ulsanin, as is readily seen from references (Deutsche Med.

Wochenschrift, p. 1512, 1913; also Year Book of American Pharmaceutical Association, 1913, Vol. II, p. 140) gives off simultaneously nascent *oxygen* and *iodin*, when brought in contact with even minimum amounts of moisture, while iodin powder (Sulzberger) also particularly differs from ulsanin, in that not a trace of oxygen is liberated even when brought in contact with any quantity of water at high temperature.

The earliest references to this German compound that I could find in literature was 1912 (Deutsche Med. Wochenschrift), showing that the statement made that such a remedy was in use for several decades is also incorrect. The party, furthermore, in referring to a substitute preparation, speaks of anhydrous boric acid. It would seem by this is meant metaboric acid; that is, boric acid which has split off chemically a molecule of water. Also the expression, "exceptionally free from water of crystallization" is not clear as boric acid has *no* water of crystallization. Evidently the above statements may have been made for an obvious reason. An attempt is being made to substitute a powder, an imitation of iodin powder (Sulzberger). It would seem that it would have been part of wisdom and more in line with scientific methods for any indivdiual to better inform himself before making statements at a meeting of any prominent medical society which can so readily be proven as incorrect by referring to available literature.

Since my name has been associated with the iodin powder (Sulzberger) treatment (the original paper appeared in the November, 1917, number of THE LARYNGOSCOPE), I feel called upon to direct attention to any statements which may be misconstrued and misunderstood and which require clarification. The splendid results observed and recorded by many colleagues and myself with this treatment, for some years, were obtained with the iodin powder prepared after the special process originated by Dr. Nathan Sulzberger, chemist of New York.

106 East 84th Street.

International Digest of Current Otolaryngology.

Editor:
Dr. Maxwell Fineberg, St. Louis.
Collaborators:
Prof. G. Bilancioni, Rome.
Mr. W. S. Daggett, London.
Priv. Doz. G. Kelemen, Budapest.
Dr. H. C. Rosenberger, Cleveland.

O. Muck, in the Klinische Wochenschrift, Berlin, March 15, 1930, presents an article on Meatless Diets for the Treatment of Ménière's disease. The author's reason is that the vasoconstrictor nasal reflex which may be produced in epilepsy and migraine may also be produced in Ménière's disease. This fact is stated to prove that the disorder is caused by angiospastic conditions in the auditory artery. Others have reported meatless diets to counteract epilepsy. This led the author to employ a meatless diet in five cases of Ménière's disease and he claims marked success with this treatment.

Curschmann, in the April 12, 1930, Klinische Woch., Berlin, reports several cases of familial nosebleeds. He could not detect thrombopenia or hemophilia in these cases. In spite of the fact that the thrombocytes showed normal behavior in all of the cases that the author examined, he assumes, nevertheless, that the nasal hemorrhages were caused by some pathologic factor in the thrombocytes. Local treatment generally failed in his cases and he recommends Roentgen irradiation of the spleen as the most effective treatment.

Schugt, in the Munchener Med. Woch., April 11, 1930, writes on the treatment of tuberculosis of the larynx by means of curative paralysis of the recurrent nerve. This method is called Leichsenring's method, which procures rest for the diseased larynx by temporarily paralyzing the recurrent nerve. This may be achieved either by injections of alcohol or by surgical intervention. Schugt used alcohol injections and employed the treatment in 25 cases; he suggests that the treatment be carried out only on one side as a bilateral paralysis might lead to suffocation.

The author claims that the rest secured for the diseased larynx is more complete and more effective than merely restraining the patient

from speaking. The average duration of the paralysis is from four to eight weeks. On the basis of his experiences, the author concludes that Leichsengring's method for exclusion of the nerve can yet be improved; he asserts that it is not dangerous and that it gives good results and, therefore, he does not hesitate in recommending its application.

The regular correspondent of the *Journal A. M. A.*, in the July 5, 1930, issue, reports a study made in Italy on children with adenoids. The census has the backing of the government and information is being recorded pertaining to the occupation of the father, economic condition of the family, the child's progress at school and his general physical condition. Some very interesting results are being obtained which show the mental retardation of children with adenoids in comparison with healthy children, and also in direct relation to the degree of hypertrophy of the adenoids.

Jellinek and Scheiber, in the April 3, 1930, Wien. Klin. Woch., discuss what they call a New Method of Hearing. They claim that the internal ear receives vibrating currents which may be electrical in character; the auditory apparatus is oscillated by electric vibrations, and hearing results. They have devised an apparatus whereby some proof of this theory is advanced.

With the meager information at our disposal at the present time, the abstractor is not in a position to comment on this theory.

DR. ROSS HALL SKILLERN.

The American Board of Otolaryngology hereby puts on record the great loss which has come to us by the death, on Sept. 20, 1930, of Dr. Ross Hall Skillern, of Philadelphia.

He was a member of the Board from its inception. He played a large part in molding its form of organization and in helping to achieve its present success. He always kept his first enthusiasm, and was untiring in carrying out the many duties which fell to him. The Board greatly profited by his personal prestige. The memory of his ability, devotion and comradeship remains as a precious asset to his fellow members.

DR. W. P. WHERRY,
Secretary.

DR. HARRIS P. MOSHER,
President.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

Meeting of March 26, 1930.

Analysis of Cases of Laryngeal Carcinoma Seen Since October, 1929. Dr. J. E. MacKenty.

Appeared in full in THE LARYNGOSCOPE, *October, 1930.*

DISCUSSION OF PAPERS BY DRS. IMPERATERI, MACKENTY AND TUCKER.

DR. SIDNEY YANKAUER: In the first place, the Section is to be congratulated upon having the opportunity to listen to an interesting and instructive presentation by Dr. Tucker and I feel that we owe him our thanks for coming here from Philadelphia to make this presentation to us.

It has always been my practice in the past to follow the accepted doctrine of removing the entire organ whenever any part of it has become affected with carcinoma. I have agreed with those who believe that a carcinoma limited to the vocal cords does not require a complete laryngectomy, but that the removal of the cord alone satisfies the above condition. However, I have limited my operative procedures to the removal of the cord alone and this I feel can be accomplished usually by suspension laryngoscopy and by thyrotomy, but the number of cases which I have seen is very small. For a number of years I have not had a suitable case until recently. Dr. Tucker, however, has been satisfied to remove only a part of the vocal cord and in the case which he last presented, which has been well for four-and-a-half years after operation, he removed parts of both vocal cords and did not remove either cord entirely. This is a departure from the accepted doctrine, but the fact that four-and-a-half years have passed since operation, without recurrence, seems to me to clinch his argument.

With regard to Dr. Imperatori's presentation, the one fact which he has dwelt upon seems to me to be worthy of more extended emphasis, and that is the importance of preserving the mucous membrane in the nasal interior. It must be remembered that the nasal mucosa is not merely the lining of the hole through which we breathe, but that it is a functionating organ having special anatomical and histological formations to exercise this function; that it cannot be satisfactorily replaced by scar tissue. In operating upon the sinuses this fact has received, to a certain degree, general recognition, because it is no longer considered good practice to ruthlessly remove mucous membrane from the frontal sinus or from the antrum, but to preserve as much of the mucous membrane as possible. Unfortunately, this attitude has not yet been extended to the ethmoid and sphenoid region, although it should be.

Chronic Subglottiditis. Dr. Rudolph Kramer.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. L. GLUSHAK: What do you do in the cases of chronic inflammation of trachea and subglottic region, especially in the atrophic type which is accompanied by dry scabs covering large areas of lumen and lower surface of vocal cord, and even encroaching over cords, as sometimes seen in conditions accompanying atrophic rhinitis and pharyngitis?

Rhinoscleroma with Laryngeal Involvement. Dr. G. Allen Robinson.

Rhinoscleroma is a chronic infectious granuloma first recognized by Hebra in 1870, although the causative agent, the bacillus of Frisch, was not discovered until 1882. This disease is especially common among the Russians and Austrians, but it does occur in other European countries. This disease usually has its origin in the nasal cavity and gradually extends to the nasopharynx and the pharynx. Rarely is it primary in the larynx.

The case presented here showed an extensive invasion of the upper air passages, including the larynx.

In Wolkowitch's series of 87 cases, the larynx was involved in five cases. In Streit's series, five out of 38 cases showed laryngeal invasion. In my series of seven cases, only one shows definite involvement of the larynx. Three early cases localized to the nasal cavity are clinically well more than four years following radium treatment.

Case Report: Mr. J. B., age 34 years, born in Russia, has lived in New York for 17 years. He first complained of nasal obstruction about 10 years ago. In all probability, however, the inflammatory process had existed before immigration to this country.

Various operative procedures have been done upon the nose and pharynx, followed by three radium treatments to the nasopharynx eight years ago.

Patient was admitted to the New York Post-Graduate Hospital in December, 1929, complaining of nasal obstruction, hoarseness, cough and dyspnea.

Examination revealed an undernourished young man with nasal cavity filled with dried crusts and stenosis in posterior nares, the uvula missing, pharynx extensively sclerosed, soft palate thickened, epiglottis retracted, arytenoids fixed, the vocal cords enveloped in the fibrous process of the slightly ulcerated thickened laryngeal walls so that only a narrow slit was available for breathing.

We have been unable to determine the amount of invasion in the trachea and bronchial tubes. Tissue removed for pathological examination revealed typical granulomas and the fungus lesions.

Three external radium packs have been applied to the larynx at 3 c.m. distance, Dec. 24, 1929, amounting to 12,000 m.g. hours. There appears to be slightly less stenosis in the larynx and, symptomatically, patient has improved. It is hoped that further pallative treatments may be obtained by radium packs in order to postpone the eventual treacheotomy.

Presentation of Cases. Dr. David H. Jones.

DISCUSSION.

DR. LEWALD: In Dr. Jones' differential diagnosis between tuberculosis, carcinoma and syphilis, he calls attention to the use of the X-ray of the chest. I wish to confirm what Dr. Jones has said, but would add a word of caution in regard to the danger of *overinterpretation of pulmonary tuberculosis*, based solely on heavy root shadows and calcified glands. This appearance is found in practically all individuals except for races in remote portions of the world, such as the Eskimos or the inahbitants of Central Africa. I have examined the X-ray findings in several thousand cases referred to the Department of Health for admission to Tuberculosis Sanitoria and was surprised to find that in about 20 per cent of the cases regarded as tuberculous I found only the shadows in the chest which resulted from childhood lesions, without any evidence of activity. In all of these cases the sputum was negative. The application of Dr. Jones' paper refers to the fact that a positive diagnosis of pulmonary tuberculosis would confirm a questionable diagnosis of tuberculosis of the larynx. If the chest diagnosis is based upon definite evidence of *parenchymal infiltration of the lung*, especially apical involvement, this evidence becomes of *postive value*. If, however, the diagnosis of pulmonary tuberculosis is based solely upon root shadows and healed childhood lesions, an erroneous opinion may be expressed, which would lead to believing that a laryngeal lesion was tuberculous, while, as a matter of fact, it might be due to an early carcinoma, and thus operative. By the time that the mistake had been corrected, the lesion might have become inoperable.

DR. L. GLUSHAK: Why do you X-ray to the larynx? What do you mean to accomplish with X-rays on keratosis?

DR. G. A. ROBINSON: Tissue removal for study microscopically is of great importance in order to determine the type of neoplasm present. Although the majority of cases will show squamous-celled carcinoma, a great many are of the basal and transitional types. The latter two lesions are radiosensitive and good results have been obtained by the use of radium, obviating the necessity for radical surgery. Biopsy also reveals the rarer tumors as foreign body granulomas and the fungus lesions.

Case Report: Laryngectomy for Carcinoma of the Larynx and Thyroid Gland. Dr. M. S. Bender.

A man, age 38 years, admitted to the medical service of Mt. Sinai Hospital, Dec. 9, 1929, because of productive cough, loss of 15 pounds in weight and general weakness. No history of illness until two weeks prior to admission, except a tonsillectomy one year ago as a promised cure for hoarseness, which was of 15 years' duration. The admission complaint was precipitated by a so-called acute cold.

Examination: An undernourished male, coughing with a mucopurulent sputum, slight cyanoses of the lips, respirations very noisy and accompanied by a distinct inspiratory and expiratory stridor, the voice distinctly hoarse. Laryngeal examination, a stenosing bilateral infiltration with a marked acute edematous congestion of the glottic and subglottic space.

Blood, Wassermann negative; sputum, negative T. B. C., Dec. 9, 1929.

Progress: The patient transferred to laryngological service because of increased progressive laryngeal embarrassment.

Tracheotomy, Dec. 13: During the tracheotomy, a small glandular nodule was excised from the region of the cricothyroid membrane. The section was later reported as being carcinoma of the squamous cell type. It was therefore felt that we were dealing with a carcinoma of the larynx.

Laryngectomy, Dec. 20: A classical MacKenty operation, under novocain anesthesia, with a T-shaped skin incision exposing the larynx and trachea. At this juncture, the thyroid gland was found infiltrated with carcinomatous tissue. It was therefore deemed advisable to extirpate the larynx and thyroid gland, *en masse*.

Patient, after an uneventful postoperative course, was discharged from the hospital, Feb. 5, 1930.

Comments: This case is presented because of the unusual type of extrinsic carcinoma which infiltrated the thyroid gland. The present well-being of the patient is an encouraging reward for undertaking an apparently hopeless procedure. It is interesting to note that this young adult, who had been hoarse for 15 years, had a tonsillectomy performed to cure his hoarseness. It might also be noted and perhaps left for conjecture, whether or not a carcinoma of the larynx existed at the time of tonsillectomy.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

Meeting of April 12, 1930.

Varicose Aneurism of the Auricle. Dr. F. W. Graef.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. A. NIGRO: Mrs. S., age 44 years, had O. M. P. T. C., right, since age 4 years, but with very little discharge. During all this time, the ear would be dry, according to her story, for months and then run a little. Examination reveals the whole mastoid pretty extensively gutted out. The lower part of the drum is intact. The posterior canal wall is eaten down and has a slight polyp on its suface. The attic has been completely eaten out. Left ear, partial deafness; hears from affected ear. Endocrine dist.

Question of Operation—Hearing.: H. B., age 32 years, right ear discharging off and on for years. At times, very purulent, creamy pus; at other times, thin and watery. Examination reveals the mastoid process exenterated, middle ear very slightly affected; hearing remarkably good; granulation tissue pressure on posterior wall of mastoid process.

Several others have been seen and are of interest to show what Nature can do.

J. B., age 6 years, parostitis; about five days after onset, seized with marked dizziness, so much so that he kept his face buried in the pillow. Examination revealed a typical nerve deafness of the right acoustic nerve with an irritative

lesion of the vestibular apparatus, followed by total loss of function of the nerve.

The classical nystagmus gradually subsided and now, two years afterward, he has entirely dead labyrinth on this side; complete nerve deafness.

Sinus Thrombosis with Pyemia. Dr. C. M. Griffith.

The case about to be presented was a white male, age 15 years, who was admitted to the Manhattan Eye, Ear and Throat Hospital, Dec. 27, 1929, complaining of pain and discharge from the left ear.

Family History: The family history was unimportant. Past History: "Rheumatism" at 4 years of age; pneumonia at six; scarlet fever without ear complications at eight; otitis media at 10. Does not remember which ear was involved. No ear symptoms since.

Present Illness: Two days before admission, developed a severe pain in left ear, which persisted about one day and was then only partially relieved by a spontaneous discharge of pus. As the pain had not subsided, he was brought to the hospital, where the house surgeon advised admittance.

Examination on admission was as follows: Temperature, 102.6°; pulse, 100; respiration, 20; heart and lung extremities normal. Profuse purulent discharge from left ear, slight tenderness over mastoid.

On the following day (Sunday), the temperature ranged from 101.4° to 104°. The discharge was unchanged, but tenderness was more marked. The blood count: R. B. C., 4,000,000; hmgb., 76 per cent; W. B. C., 24,000; P. M. N., 88 per cent. The patient appeared toxic and in the afternoon suddenly complained of a pain in the right knee. Examination later in the day disclosed the above findings and also a markedly swollen and tender right knee joint. The medical consultant was of the opinion that it was a metastatic process and therefore immediate operation was decided upon.

Dec. 29, 1930: About four days after onset of the acute otitis, the mastoid was opened and found to be extensively involved and contained much free pus. There was no pathological exposure of sinus or dura. After thorough exenteration of the cells, the lateral sinus was uncovered and inspected. There was no gross evidence of involvement.

The sinus was then opened widely and free bleeding noted above and below—after packing of the sinus—the jugular vein was ligated above its facial tributary. On the day following operation, the temperature remained at 103° to 104° and the knee seemed worse; blood culture started before operation was negative.

Dr. Phelps was called in consultation and on Dec. 30 aspirated the knee, obtaining a purulent fluid (which when cultured showed no growth); he then advised that the patient be transferred to another hospital for arthrotomy. Due to an unfortunate misunderstanding, the patient was not immediately transferred, remaining in the ward two more days. However, his general condition was better.

Jan. 3, 1930: Transferred to St. Mark's Hospital, from whose records the following notes were taken: On admission, temperature was 99°, right knee swollen, extremely tender and fluctuating.

Operation, Jan. 4: Lateral incisions both sides of the patella, about 4 oz. of pus evacuated. Following operation, the temperature remained normal until Jan. 9, when there was a rise to 103° and evidence of further infection around the joint. From Jan. 9 to Jan. 16, the temperature was of the septic type, ranging from 99° in the mornings to 104° in the afternoons.

Jan. 16: Incision and drainage of infected hematoma of right knee. Pus had extended upward, downward toward head of tibia and back into popliteal space. In spite of this further incision, the process progressed.

Jan. 24: Original incisions extended, new incisions made; patient's condition poor.

Jan. 30: Transfusion, 500 c.c.. In spite of these efforts, the infection continued to spread and it was decided that amputation was necessary.

Feb. 6: Right leg amputated at mid-thigh. Examination of amputated member showed complete destruction of the joint; abscess halfway down the shaft of tibia.

Feb. 6: Blood transfusion, 500 c.c. Following this last operation, there was a slow but uneventful convalescence and the patient was discharged, March 9, as cured.

Smears and cultures from the knee joint disclosed a Gram positive coccus in diploid and short-chain formation. The blood culture was always negative, but unfortunately the mastoid culture was lost and there is no record of the organism found.

The case seems interesting: 1. Because of the rapidity of the process (a purulent arthritis four days after onset of an acute otitis media. 2. The severity of the knee infection, necessitating amputation. 3. Absence of any other metastasis. 4. The bacteriology, probably the strep. mucosus. capsulatus.

DISCUSSION.

DR. RALPH ALMOUR: Dr. Griffiths has presented a very interesting case. I am only sorry that no culture of the mastoidal suppuration was obtained, so that it might have been correlated with that taken from the knee joint.

I have seen several similar cases, which, within a few days after the aural infection, showed a marked destruction of cellular elements within the mastoid process. The cases I allude to all presented, simultaneous with ear lesion, an infection somewhere else in the body. Three showed an acute hemorrhagic nephritis, one having an albuminuric retinitis. Another showed bronchopneumonia.

My conception of these cases is that there existed a focus of infection in the nasopharynx which infected the two different organs at the same time. I feel that the case reported by Dr. Griffiths fits into this group and that the knee was infected simultaneously with the ear. With the type of pathology found in the mastoid bone, typical of the coalescent mastoiditis, one would expect a sinus thrombosis to be preceded by a perisinal abscess. Such was not the case here. The sinus itself was normal in appearance and, when opened, showed no evidence of a thrombus. Furthermore, there never was any sepsis nor any positive blood culture. I am therefore inclined to believe that the joint lesion was metastatic, not from the ear, but from some other source, probably the nasopharynx, which infected the ear and joint at the same time.

DR. DAVID SHAPIRO: I thought the knee infection came after the operation, and wondered why he opened the sinus, and whether the temperature was septic when he operated. I wondered why he did not operate simply on the mastoid and then wait for further indications before operating on the sinus.

DR. GRIFFITHS (closing): It is true that no pus was demonstrated in the sinus, but at times if we wait to demonstrate pus we may lose the patient. Opening the sinus is not going to do any harm, and the assumption is to infection of the mastoid. Of the cases we see at the Manhattan, many run a high temperature, not the classical up and down temperature. I would not think of doing a mastoid and not opening the sinus in such a case, where there were focal symptoms elsewhere. If there is any chance of saving the patient, you will not lose anything by doing it.

Auditory Nerve Deafness. Dr. A. Schattner.

Appears in full in this issue.

DISCUSSION.

DR. E. M. JOSEPHSON: The title and subject of the paper is one which immediately raises a question of definition, which I am afraid Dr. Schattner has not answered. A glance at our textbooks will show how much difference of opinion there is with regard to what nerve deafness is, especially from the clinical angle.

It is hard to understand how one can speak of nerve deafness so long as bone conduction be present; for, whether the sensation of hearing comes by air or bone, the impulses must travel by way of the auditory nerve and so long as the impulses are carried by the nerve, *via* bone or air, it cannot be regarded as destroyed. It is far more rational to speak of nerve deafness in those cases in which the pathologic pictures of destruction of nerve endings in the organ of Corti over extensive or limited ranges are paralleled clinically by extensive or total, or by limited defects of both bone and air conduction.

Contrary to the belief widely adhered to, the auditory nerve, like nerve tissue in general, is comparatively resistant to disease process and injury, and

has a truly remarkable power of repair and recovery. The ever increasing number of cases of recovery of hearing after labyrinthitis and labyrinthotomy prove this recuperative power of the nervous mechanism of the ear. But where the blood supply of the nerve is cut off or reduced over a long period of time, the auditory nerve, like other nervous structures, is generally permanently damaged. All evidence indicates that one must look to the vulnerability and lack of reparative power of the vascular system for a full appreciation of the etiology of deafness and other nerve tissue damage.

In this connection it might be mentioned that there has been advanced a number of hypotheses in regard to the relation of vascularity with vestibular function. Dr. Whitham, in addressing this Section several months ago, stated that the validity of the theory that vestibular function was dependent upon changes in vascularity could be proved by determining the response of the vestibule to drugs which directly affect blood supply and vascularity. This has been done. It has been found that applying adrenalin to an otherwise fairly normal middle ear, one causes a drop of hearing and a rise of vestibular irritability. Conversely, using vasodilators, one gets a rise of hearing and a drop of vestibular irritability. That there be some relationship between vestibular and auditory function, we might very well have expected *a priori* did we stop to consider the intimate relationships of the cochlear and vestibular apparatuses.

The relation of vascularity to hearing in the cases of supposed nerve deafness, using the term, in absence of a definition of the exact type of deafness which Dr. Schattner has discussed, as synonymous with a certain type of chronic progressive deafness, has been noted in the literature by Dr. Albert Gray and myself. One finds that vasodilators will occasion a momentary rise of hearing in many of these cases of incomplete deafening.

I have made some further studies than those which I have published, on the relation of ear vascularity to hearing with the aid of a device which permits of viewing the vascularity of visible ear structures, magnified 20 to 40 times. Findings made by naked eye, published last year, have been confirmed and an interesting light has been cast upon that form of supposed nerve degeneration and deafness, which Dr. Schattner classifies with the nerve deafnesses, known as senile deafness.

I had for some time been impressed with the parallelism of vascular and myocardial tone with hearing in senile cases. The hearing of these cases, which has been extensively studied by Dr. Bunch, generally shows a depreciation and loss of the upper range of tones. I have always felt that there is a direct causal relationship between the vascular status of these cases and their hearing. With the aid of the microscope, I have been able to demonstrate the impairment of blood flow in the capillaries of the ear drum and of the external auditory canal in these cases. When these cases are digitalized and their general circulatory condition improves, the capillary circulation of the ear improves and, paralleling this improvement, the hearing improves, sometimes returning to approximately normal.

As regards the difference of involvement of vestibular function in the congenital and acquired forms of nerve deafness, it has been my experience that one fails to find vestibular involvement in few of the cases, if the more delicate tests of vestibular function be used. We have been in the habit of using the cruder tests of response of the vestibular apparatus to forms of irritation rather than the responses to the normal forms of stimulation, such as the oculo-vestibular past pointing tests, the umbrella tests, the galvanic tests and others. For those congenital cases where there appears to be a minimal involvement of the vestibular involvement with a maximal involvement of the cochlea, it is most reasonable to assume failure of ochlear development; for it is hard to conceive that a disease or degenerative process could selectively strike one of two organs so intimately interrelated structurally. These cases do bear out the antithetic responses of the vestibular and cochlear apparatus to variations in vascular balance of the labyrinth, which has been mentioned.

DR. RALPH ALMOUR: There can be only one true classification of deafness, and that one is based on pathology. Any other type of classification, particularly a clinical one, is provocative of discussion which is irrelevant to the

subject presented, since the clinical findings vary to such an extent and are capable of many and varied interpretations.

The auditory apparatus can be involved in its perceptors, its terminal ganglion, the nerve trunk proper, or in its nuclear and supranuclear pathways. Drugs, chemicals and certain toxic products of metabolism affect the cells of the spiral ganglion. Epidemic meningitis rarely destroys hearing by an extension of the infection through the internal auditory meatus. Where such occurs, a suppurative labyrinthitis results. As Wittmaack, Alexander, Eckert-Mobins, Manasse and others have shown, the deafness following meningitis is the result of toxic products which find their way through the aqueductus cochlearis into the perilymphatic space and set up a serofibrinous labyrinthitis confined to this space. This in turn sets up a hydrops of the endolymphatic space which, by its increased pressure alone, causes a greater or lesser destruction of the organ of Corti. The lesion is healed by a metaplastic ossification.

Dr. Schattner has stated that cases of congenital nerve deafness show the presence of labyrinthine function, whereas cases of acquired nerve deafness usually do not respond to the usual functional tests. I cannot agree with this statement as I have seen numerous cases of the acquired type wherein labyrinthine function was present and wherein normal responses were obtained.

DR. SCHATTNER (closing): Before making any remarks, I wish to thank the gentlemen for their kind interest in my paper. When I wrote it, I was informed that I should limit myself to 15 minutes, and it is clear that a subject of this scope cannot be presented completely in that space of time.

As far as the classification is concerned, I must differ with Dr. Almour about the pathological classification for the clinician, because it is not always possible to make a pathological diagnosis; moreover, many similar pathological conditions will give a different clinical picture; thereby giving no indication as to the mode of treatment to be pursued; and for this reason Alexander devised his classification. I think the pathological classification is not of great value to the clinician. Of course, there is still some question about the proper classification; I do not think we have that yet; I have only chosen Alexander's because it seemed to be the most suitable for the presentation of the subject.

I stated in my paper that acquired deafness involves both the cochlear and the vestibular mechanism; I must stand by that statement. In congenital internal ear involvement it is usually the inferior ramus which is involved; that is, the cochlea and sacculus.

This statement is in accord with the reports of ear involvement observed in cerebrospinal meningitis cases and contradicts Dr. Almour's statement that frequently normal labyrinthine responses are obtained. I must also stand by my statement that the process by which the eighth nerve is involved in cerebrospinal meningitis is usually a direct invasion along the nerve sheaths through the internal auditory meatus leading to rapid destruction of the end organs. Alexander considers this the usual route and the hematogenous route the exception. I must refute Dr. Almour's statement that there are no greater communication between labyrinth and endocranium in the infant than in the adult. In the infant, the aqueductus cochleae is very short and wide, as is also the meatus acusticus internus, whose diameter may be even greater than that of the adult, as shown by Alexander. The external surface of the dura is more intimately connected with the cranial surface of the petrous bone, and a dural process in the fossa subarcuata penetrates deeply into the petrous bone.

Dr. Josephson's statements on senile deafness were very much to the point. I just made the statement that these degenerative conditions were found. I do not believe that it is a closed issue as to whether they are due to toxic action or to a circulatory disturbance.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Regular Meeting, Tuesday, April 1, 1930.

Nontraumatic Cerebrospinal Rhinorrhea Accompanying Acromegaly. Dr. Bennett F. Roberts.

Summary: 1. Central nervous system disturbance first noted in 1916 along with hypophyseal changes and eye changes. 2. Frequent peritonsillar abscesses since adolescence. 3. Typical eye as well as X-ray findings of a chiasmal lesion. 4. Three years previous to the onset of the rhinorrhea, in 1929, her nervous symptoms had apparently cleared up to some extent. 5. Since May, 1929, there has been a constant clear, watery discharge from left nostril, occurring at times in a gush and at other times in a constant dripping, ranging from 10 to 20 drops a minute for 10-minute periods, eight or 10 times a day. In October, 1929, the discharge ceased for six weeks, during which time she suffered another attack of peritonsillar abscess and was confined to her bed for several days. Conversation with patient on March 29, 1930, revealed that her nasal discharge had increased and that her headache had returned.

X-ray report, May, 1917: Tumor in region of pituitary which has extended through the floor of the sella and invaded the left sphenoid; the clinoid processes intact. May, 1930: The clinoid processes are destroyed. The tumor has extended through the floor of the left sphenoid.

DISCUSSION.

Dr. Loftus: I think that this is a very unusual paper, because of the fewness of the cases. Should this case come to a fatal issue (as the majority of the cases do), I sincerely hope that you will have the opportunity of doing a necropsy. I reported a very similar case in 1922, but unfortunately did not have the opportunity of posting it. Although we had the privilege the pathologist did not arrive at the stated time. In this particular patient of mine I was very suspicious of pituitary disturbance, but the studies of Dr. Riesman, Dr. McCarthy and Dr. Pancoast did not corroborate this. This woman had some of the characteristics of pituitary dysfunction; she weighed 240 pounds, had a heavy growth of hair on her face and body.

This case was studied very carefully from every angle, and I also studied every case published up to the time of the reading of my paper. Never before the reading of my paper had anyone thought that cerebrospinal rhinorrhea may be caused by a congenital defect remaining in the base of the skull, namely: the craniopharyngeal canal. Leber regarded his case as undoubtedly one of hydrocephalus internus, in which the ossification of the skull lead to increased intracranial pressure. In other cases to which he was able to refer, which were reported by Thomson at the date of Leber's publication (1883), he ascribes to a late hydrocephalus, although he allowed there was no direct evidence of this. Other factors which point toward hydrocephalus in contradistinction to tumor are the long duration of the illness in most cases and the constant absence of symptoms to one locality. Up to the time of the reading of my paper only four cases had a necropsy performed. These cases were reported by Gutche, Mermod, Paget and Baxter. Gutche's case is of value in a negative way, as it shows the absence of localized disease. In Merod and Paget's case the patient died from traumatic infection of the meninges, which complicated the pathologic picture. The necropsy findings in Baxter's case were negative. It is interesting to note that the necropsy findings in these cases did not support the theory of Leber that cerebrospinal rhinorrhea is due to hydrocephalus internus.

In the majority of the reported cases the flow was from the left side; in a few cases it alternated. In only one case reported the flow came from both sides. This, however, can be questioned, since it was not accurately determined. I was never able to determine the exact site of exit of this fluid. It always came from the left side, except when I firmly packed the left naris; it then

escaped just as freely from the right side. These cases should be studied carefully because of their rarity.

If Dr. Roberts has an opportunity of studying this case more thoroughly, I would like to offer a suggestion of doing a ventricular puncture and to inject either air or a dye to determine size of ventricles. I sent my patient to Dr. George P. Muller for such a study, but when she learned that it was necessary to insert probes into her brain, she positively refused to allow any surgical intervention.

DR. COATES: My only experience with spontaneous cerebrospinal rhinorrhea is in this particular case that Dr. Roberts has so well and interestingly reported to you. Very few cases of spontaneous rhinorrhea have been reported in the literature and we have been able to find none connected with acromegaly, although it has been noted as being associated with pituitary tumor. I feel confident that there must have been many more that had been overlooked and therefore not reported, but since it is evidently a comparatively rare condition, it will be interesting if any of you who run across such cases would study them and write them up.

As for traumatic cerebrospinal rhinorrhea cases, that is another matter. Undoubtedly, many of these have occurred following intranasal operations, but operators, as a rule, are a bit modest about reporting them. Some have done so, however, and made very good reports.

This woman is a typical case of acromegaly with massive bones of the head, big hands and feet. She also complains a good deal of headache. Dr. Roberts has not completed his studies because she has evidently become a bit alarmed at being made the subject of a report and has been hard to handle. We hope, however, soon to be able to persuade her to come back in order to have a check up by X-ray and, in particular, to study her fields of vision.

DR. WIEDER: I would like to ask a question. What would be the harm of operating on her for pituitary disturbance if evidently from description of the X-ray and history of the case, the pituitary has penetrated the sphenoid? Since they can operate on pituitary tumors, by the Hirsch method, without being followed by cerebral spinal fluid discharge, possibly if they got rid of the tumor, the rent in the dura would close up and the danger of infection avoided.

DR. BRICKER: Two years ago a case similar to this came to my attention. It was a very unsatisfactory case to follow. This boy was age 20 years, having been one of the youngest graduates at the Philadelphia College of Pharmacy. He was very effeminate in appearance. His breasts were well developed and he was very delicate. Diseased tonsils were removed several years before this condition developed. He developed dripping from the nose. This kept up for several years, when he developed meningitis and died.

Saccharomycosis of the Palate. Dr. Henry A. Laessle.

Published in full in THE LARYNGOSCOPE, *September, 1930.*

DISCUSSION.

DR. STOUT: Did I understand that this sore mouth was the same as that of aphthous stomatitis which attacks children so commonly? If it is, I would suggest that you try a preparation containing resorcin, borax, alcohol and glycerin properly diluted with water. I have used this many times in the past and have not known it to fail. However, it seems that we are developing new infections which have manifestations in the mouth, although this condition of the mouth is secondary, while the general condition is the serious part of it. I refer to agranulocytic angina, mononucleosis and trench mouth. I have seen two cases of mononculeosis, one just recently, which started with a sore throat and had a small white patch over the tonsil area. This responded at once to 20 per cent silver nitrate but he developed at once cervical adenitis, painful swallowing, pansinusitis, bilateral otitis media going on to mastoiditis which had to be operated upon. He finally recovered and I hope to show the patient at some later meeting of this Society. The question is, are these new diseases or are they manifestations of the same diseases, the difficulty being that our patients lack resistance.

DR. YAZUJIAN: Some years ago we had a very interesting case of fungus disease at Mercer Hospital. The patient had been in the medical ward with an obscure lung condition. She had been expectorating large quantities of grayish-brown pus. Her case was considered hopeless by the medical men on duty. Ulcers had developed in the eyelids, for which condition she was referred to the eye service. We found that the pus in the lid ulcers had the same color and consistency as the pus in the sputum. Made a tentative diagnosis of fungus infection and suggested large doses of potassium iodid. The microscope and the therapeutic test confirmed the diagnosis. The ulcers in the lids and the lungs cleared up in a few days and the woman was discharged as cured at the end of a week.

DR. LAESSLE (closing): Dr. Weider asked if this monilia was a recent growth and how long standing was the perforation of the septum and of the hard palate.

Dr. Stout suggests resorbin to eradicate the monilia, and that in former years mothers used borax, glycerin and honey with good results.

Dr. Yazujian relates the use of potassium iodid internally is of value.

In answer to Dr. Weider's question, will state that the growth of the monilia has been of four to five months' standing and, from the history of the patient, have been unable to get a definite history as to the duration of the perforation of septum and of the hard palate. In spite of the negative findings of Wassermann and Kahn reactions, the skin clinic diagnosed it as congenital lues. As for the growth, we find that the monilia grows best on beef culture media, which is slightly acid in reaction.

Dr. Stout's question: I did not use resorbin. While the former mothers used borax, which is alkaline in reaction, glycerin being slightly acid and really belongs to the alcohol series, the trihydric group, the honey and glycerin act as a vehicle for the borax, hence we readily understand how this mixture of alkaline reaction would inhibit the growth of monilia.

Dr. Yazujian's suggestion of potassium iodid treatment is well taken. Castellani reports that the cases in tropical and subtropical regions, especially where the growth involves the bronchus and bronchi, potassium iodid is administered with fair results.

Laboratory Studies in 150 Cases of Stammerers. Dr. Max Trumper and Dr. Robert M. Lukens.

Published in full in THE LARYNGOSCOPE, *September, 1930.*

DISCUSSION.

MRS. STEEL: I came here tonight to learn of the results of Dr. Trumper's laboratory tests and not to speak, for my work is the practical correction of defects of speech. The cases are referred to me by members of this Association for speech training.

The fact that Dr. Trumper's cases had all rested before the tests and registered these deviations from correct breathing is most interesting, but I am wondering whether the same tests made with the same number of nonstammerers would not have shown as great a variety of deviations.

I follow the teaching of Dr. G. Hudson-Makuen and believe that we have two kinds of breathing: passive breathing, shown in Dr. Trumper's pictures; and active breathing, used for voice production. Good speech is dependent upon three peripheral mechanisms: breath, voice and articulation; and these three must work with the mind. Stammering is a psychophysical inco-ordination. Some stammerers sing well, but in speaking they lose the auditory picture of the word which they wish to speak, because fear of something blots out the tone image which makes the vocal cord vibrate. The stammerer loses the image of the tone and fear makes his breathing irregular.

I am quite sure that nasal obstructions—tonsils and adenoids—are very often, as in the cases reported by Dr. Lukens, the cause of defective breathing and speech. I am glad to have had the privilege of seeing Dr. Trumper's pictures and I hope that similar tests will be made with nonstammerers and of active breathing during voice production.

DR. STOUT: A shock may bring on stammering, and it may also stop it. I believe there are two parts to our brains that have to do with speech: one a memorizing part, and one for the control of our usual speech. One who stam-

mers and stutters may be able to recite yards of poetry, which he has memorized, without stuttering. On the other hand, if he begins to speak, he will stammer and stutter. I know of others who stammer and stutter when they begin to make a speech but after they are started they may be able to deliver a great oration without any sign of impediment.

DR. BRICKER: I have had several conversations with neurologists and they have a new theory that the conversion of left persons to the use of the opposite limb seems to produce a bombardment of impulses and develops either stuttering or stammering. Dr. Winkelman is quite positive about this theory.

DR. TRUMPER (closing): I purposely made no attempt to correlate the laboratory findings and the clinical classification. If the laboratory worker classifies the patient before he makes a test he may become biased and unconsciously allow the tentative diagnosis to influence his findings. I do want to add a few remarks: Dr. Lukens referred to the occurrence of laryngeal anesthesia in approximately one-third of the patients studied. This finding is of interest in view of the work of Hugo Stern, who found many vagotonic phenomena in the majority of stammerers whom he examined. Dr. S. T. Gordy, who examined a number of these patients for us, likewise noted that many fell into the group clinically termed vagotonic, that is, they showed symptom complexes referable to disturbances of the vegetative nervous system.

Regular Meeting, Tuesday, May 6, 1930.

The May meeting was devoted exclusively to papers and discussions by the graduating class in otolaryngology, Graduate School of Medicine, University of Pennsylvania. In only two or three instances were the discussions carried on by members of the faculty in cases wherein they were particularly concerned. The cases and the material in the papers utilized were those which came within the experience of the men within the school term or cases of their own practice previous to their entrance into the class. The latter were discussed in the light of their newer and additional knowledge and experience gained.

Agranulocytic Angina. Dr. Harry Horn.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. EMANUAL SIMON: Dr. Horn has practically covered this somewhat rare condition and I wish to only bring out a point that one gets from reading numerous case reports and that has been frequently expressed this evening and in the last two years, namely, that this disease picture is not an entity.

First, one must remember that it is not only the granular cells that are reduced but that the lymphocytes are decreased as well. Normal blood has 2,000 to 3,000 lymphocytes per c.m.m. Patients with this illness usually run a white count averaging about 1,000 cells per c.m.m., and of these, 90 or even 100 per cent are lymphocytes. That would show at most 1,000 lymphocytes per c.m.m. of blood and, as we all know, the polymorphous cell is less resistant to destruction and therefore destroyed in greater number.

Secondly, this disease has been associated with unusually minor incidents in acute cases, and then again has followed protracted illnesses. The acute onset has followed such incidences as removal of teeth, typhoid inoculation, arsphenamin which previously had been causing mild toxic symptoms, fracture of bones, severe emotional upset and many other minor ailments, but usually in individuals who were in poor physical health.

The symptoms in this disease are unusually varied and the only distinctive criteria are a leukopenia, body ulcerations and prostration, often inconsistent with clinical findings. This disease is seen in certain cases of sepsis and Hodgkin's disease. Schultz in his last paper on this disease in the Munchen Medical Wochenschreft thinks this is a disease entity and due to an infectious or toxic agent destroying the myeocytic system. Others insisted that fatal arsenic poisoning closely resembles the disease.

Finally, this is a disease entity for we see this leukopenia in severe prostration and in many terminal infections of varied causes.

DR. SHUNJI KAY IKUTA: Due to lack of time, only the salient features are presented. Mine is more a presentation of two cases of agranulocytic angina observed when in Los Angeles, Calif., rather than a discussion of the paper. The cases to be presented are atypical as far as the age is concerned. Usually agranulocytic angina occurs in middle-aged women, 30 to 50 years. The proportion of female to male is given as 3.5 is to 1. Case 1: Miss K., age 20 years. Case 2: Mr. P. R., age 70 years, depicting somewhat the extreme of age. Sex was equally divided here.

The diagnosis was made chiefly by the laboratory findings, checked and double-checked by physical examination, plus autopsy findings.

History, Case 1: Miss K., age 20 years; student. Entrance complaint: Sore throat, fever and prostration. Physical examination: Marked angina, tender, enlarged cervical glands, anemia, rash and purpurgic area over extremities and slightly enlarged spleen. Laboratory report: Blood: Red, 2,760,000; hemoglobin, 48 per cent; whites, 650; 100 per cent small lymph.

Case 2: Mr. P. R., age 70 years; occupation, miner. History: Entrance complaint, second degree burn of feet. Physical examination: Second degree burn of both feet. Nose and throat examination negative.

Laboratory report: Hemoglobin, 100 percent (Sahli); W.B.C., 23,000; polys., 82 per cent; lymphs., 18 per cent.

About 1½ months after entrance, patient suddenly developed a temperature of 101°. P.X. showed marked redness and inflammation of throat and a white-greenish exudate high in left tonsillar fossa. W.B.C., 3,300; lymphs., 100 per cent. Question of agranuloyctic angina.

Blood count two days later: W.B.C., 3,000; lymphs., 100 per cent. Symptoms and throat findings exaggerated.

Blood count day of death: W.B.C., 900; lymphs., 100 per cent.

The county coroner's autopsy report read for Case 2: Cause of death, agranulocytic angina. Contributory factor, second degree burns of feet. Other findings: Jaundice of skin; heart and lungs negative; liver, gall bladder and kidneys not diseased; spleen moderately enlarged and firm; white, pinhead-sized spots on capsule.

Summary of the two cases showed the following: Somewhat extreme of age, sex equally divided. Cause: Onset of angina in both cases was sudden. Course of the disease rapid. Cause of agranulocytic angina undetermined or not known. Prognosis fatal in both cases. Miss K., two days after onset of angina; Mr. P. R., six days after onset of angina. Treatments instituted were of no avail, blood transfusion, leukocytic extract, neo, glucose saline intravenously and symptomatic RX given. Diagnosis was made by the angina, leukopenia, agranulocytic and fatal outcome; double-checked by autopsy findings.

DR. HERMAN B. COHEN: There is really nothing left for me to speak about as the subject has been so thoroughly covered by the essayist and other discussors.

Dr. Zikowski, of Vienna, described this ailment as "a severe infection in a weakened organism. That there is a paralysis of the liver, spleen and endocrine organ complex, which disturbance does not permit the secretion of these organs to regulate and release the granular cells into the bloodstream."

In time, I feel, this disease, if it does become an entity, will be classified with the blood dyscrasias. The fact that ulcers do not only attack the mouth and throat but other parts of the body as well, namely, the vagina, vulva, anus and skin, removes this ailment as a specific disease in our field of otolaryngology.

From the different lines of treatment, X-ray, early and frequent transfusions, etc., some have gotten well, but most of the patients have died. In many cases a history of shock to the nervous system was obtained, probably suggesting sympatheticonia. Cases with allergy backgrounds have been reported. We know so little about this condition that we cannot as yet place it or klassify it properly.

Vasomotor Rhinitis. Dr. Joel J. Pressman.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. ALFRED R. ROBBINS: The term turgescent rhinorrhea as applied to this condition by Dr. Pressman is a suitable terminology describing the pathological and clinical findings. This condition is a frequently found neuromuscular or muscular phenomenon, the etiology of which has been disputed. In his recent resume of this type of phenomenon, Duke states that histamine is probably the agent within the tissues producing the reaction.

To successfully diagnose and treat the disease, it becomes necessary to determine the extracorporal factor causing histamine producton. It has been generally agreed that one or more of three factors are involved; however, there is not agreement as to their relative importance. It is recognized that bacterial proteins will produce this condition, as will the food and pollen group of proteins. Also, we have physical agents as causative factors, such as light, heat, cold and friction. Under friction or pressure can be included anatomical abnormalities of the nasal passages.

Frequently the case has more than one factor involved, but one of the factors usually outweighs the others, and its removal gives only variable relief without the removal of the other factors.

Careful history and examination are imperative. Examination of nasal secretions, including cell count, is helpful, and the finding of more than 10 per cent of eosinophiles suggests an allergic factor.

Conditions of this type require careful study, and no set rules can be applied. The examiner should bear in mind the etiological possibilities and by a process of elimination arrive at a satisfactory form of treatment.

DR. WM. K. KISTLER: In the treatment of vasomotor rhinitis it is well to remember that the etilogic factors may be allergic, nonallergic, or both. The first essential to treatment is accurate diagnosis,.and while this may be possible by the history alone, skin tests have proven of particular value in this connection.

When skin tests are employed as a guide to treatment by desensitization, it is essential to use dilution for the purpose of determining the degree of sensitization. Diagnostic tests may be conducted with the commercially prepared allergens or with the alcoholic extracts of different proteins. Those producing positive reactions should be retested, according to Wallace, with dilutions of purified proteins. Not infrequently a patient will react to more than one protein; but only that protein needed be used for desensitization which yields a positive reaction with a dilution of 1/1,000 or higher.

The treatment of the allergic type may be of two kinds: 1. The removal of the exciting cause, if this is possible. 2. Desensitization by weekly injections of an extract of the protein of proteins.

Exclusion of the exciting agent or agents is of the utmost importance and may be sufficient. When this cannot be accomplished or is incomplete, desensitization is necessary.

It is advisable to determine by quantitative skin tests the degree of sensitization and commence treatment with 0.1 c.c. of the highest dilution failing to give a positive reaction. A course of 28 to 30 injections given. This should be repeated each year for two or three years.

A special effort should be made to remove foci of infection in the accessory nasal sinuses, apices of the teeth, in the tonsils or elsewhere, if these exist and are suspected as being responsible for the sensitization.

For those patients who cannot be treated as already outlined, or in whom the desensitization treatment fails, a change of locality away from the causative protein is advisable. When avoidance of the causative protein is out of the question, as well as desirable, treatment, dark glasses, a boric acid eye wash, adrenalin nasal sprays and saline nasal douches alleviate somewhat the acute symptoms.

DR. JOHN A. MURPHY: I enjoyed the paper on vasomotor rhinitis very much. I think Dr. Pressman has covered this condition very briefly and concisely for you, and I agree with him that it is a very puzzling illness to both patient and physician. The classification, allergic and nonallergic, is probably

as satisfactory as any we have today. It is often designated as perennial rhinitis or perennial hay fever.

In reference to the diagnosis of this condition, I missed one or two familiar things, namely, the so-called red nose and the so-called pale nose. The pale mucous membrane being indicative of allergy and the red nose indicative of infection. Itching is quite characteristic of allergy. I want to emphasize the item of itching, particularly itching of the eyes. You may also get it in the nose and roof of the mouth.

I was glad to hear Dr. Pressman mention the eosinophiles. They have been bobbing into allergy for several years. First they were studied in the blood-stream. Now there is the question of discovering the eosinophiles in the nasal secretion. I have often wondered if it would not be wise sometimes to let patients take glass slides home with them and make a series of smears to be studied later. The presence of eosinophiles in any or all would aid in fixing the type or classification.

Before I sit down I would like to mention an article that I recently saw by Dr. Hyram Bird. He quotes a definition to this effect, that "a state of disease is never a thing in itself but always a quantitative change in some physiological process, an increase or diminution of something that was there to begin with."

On the basis of this definition, the symptoms of vasomotor rhinitis and hay fever are expressions of dysfunctions of the secondary, motor, respiratory and secretory mechanisms of the nose and are due to excess efferent impulses to the nose by way of the nasal nerve and Meckel's ganglion.

Treatment then consists of blocking these efferent nerve paths by injecting alcohol, first locally anesthetizing for clinical observation and to determine the extent of his subsequent injections of alcohol. He quotes one instance in which he did this work on one side, this case presenting enlarged tonsils and turbinates, with the result that the turbinates and tonsil regressed in size while the uninfected side showed no alteration. I am giving you this for what it is worth. It seems to me that the after-effects might be worse than the disease itself.

Carcinoma and Tuberculosis of Larynx. Autopsy Report of Two Cases. Dr. Chas. K. Shofstall.

Appears in full in this issue.

DISCUSSION.

DR. W. S. SNYDER, JR.: It might seem quite appropriate at this point to discuss the differential diagnosis between laryngeal tuberculosis, syphilis and malignancy. However, three minutes is not sufficient to do justice to this subject.

Dr. Shofstall mentioned the possibility of syphilis being an etiological factor in the production of carcinoma and tuberculosis of the larynx. It is true that there may be both conditions present at the same time. It is further true that syphilis will hasten the course of tuberculous laryngitis, but that syphilis could be an etiological factor has no basis as supported by clinical evidence.

The pathology of tuberculosis of the larynx is similar to the pathology of all tubercle lesions; modified by the peculiar anatomical structure of the organ. Tubercules are formed and connective tissue is stimulated. When the tissue is loose and glands are plentiful, tubercle lesions are first found. Thus the posterior portion of the larynx is most frequently attacked. The edema in the larynx may be a true edema, but most frequently it is an infiltration. In the subepithelial layers there is sound cell infiltration, connective tissue hyperplasia and typical tubercules. This infiltration may shut off the blood supply to such an extent that an ulcer may be formed, or the ulcer may be formed by caseation necrosis of tubercules near the surface. These ulcers are shallow, their edges wavy and ill defined. The base is clean, often being filled in with granulation tissue. Ulcers are most common on the vocal cords and least frequently seen on the arytenoids. Tuberculoma may form—especially in the posterior commissure. The condition may resemble papilloma and the tuberculous disease be found only in the underlying connective tssue.

The parts of the larynx involved, in the order of their frequency, are as follows: 1. The interarytenoid region; 2. the arytenoids; 3. the vocal cords; 4. the ventricular bands, and 5. the epiglottis. The larynx, along with other

mucous membrane in the tubercular, has a pale, anemic appearance. Over those areas of the larynx where the tissues are lax, *viz.*, the arytenoids, interarytenoid region and false cords, the disease manifests itself by edema and infiltration. Ulcers are seen only late in the disease. In the posterior commissure, where the tissue is quite lax, papillomatous processes may be found. The cords are attacked near the posterior commissure, generally bilateral, as the process is wont to be, ulcerates early and gives a mouse-eaten appearance. The epiglottis is usually attacked on its lingual surface. There is infiltration near the median line. This spreads until the whole of the epiglottis is thickened and deformed, sometimes giving it a "turban-shaped" appearance.

DR. CLERF: Laryngeal growths assocaited with either syphilis or pulmonary tuberculosis present difficult diagnostic problems. Errors in diagnosis will occur unless a complete study be made and a definite routine with regard to laryngeal diagnosis be adhered to. Whenever there is a question in diagnosis, biopsy is indicated and particularly is that true if cancer is suspected. This would be advisable in the case of a patient with a widesrpead laryngeal lesion typical of tuberculosis, who also had a pulmonary lesion. Syphilis has nothing to do with the etiology of cancer of the larynx; it may unfavorably influence the progress of tuberculosis.

Headaches of Nasal Origin. Dr. Leo J. Hombach.

Appeared in full in THE LARYNGOSCOPE, *October, 1930.*

DISCUSSION.

DR. J. EDWIN SCOBEE: I am sure we will agree that Dr. Hombach has covered his subject very well, considering that the field is so broad and that his time is necessarily limited.

It is quite true that headache is an almost universal symptom and may be associated with almost any type of disease, although looking for headache of nasal origin, the picture may be clouded. I have in mind a case of my own of three years ago. A young man, age 21 years, complained of constant dull headache throughout the head, especially in the occipital region, of one week duration, more marked at night, with some remission during the day, associated with slight postnasal discharge. Headache only partially relieved with asperin. History and general examination showed the patient to have chronic constipation, and urine showed heavy amount of albumin. Complained of dull ache over the kidneys but no other signs of renal trouble. Nasal examination showed poor aeration, turbinates enlarged and congested, and a deviated and thickened septum. No pus was seen anteriorly and the sphenoid ostium was not visualized. There was a thick, adherent postnasal discharge coming down the lateral aspect of the pharynx. In view of the medical findings patient was given treatment for constipation and for his nephritic condition, together with some drops for the nose. Two weeks showed no improvement and patient finally consented to an X-ray of the sinuses as a possible focus of infection. This revealed cloudy sphenoid and posterior ethmoid cells. Irrigation of the nose and washing of the sphenoid did not help the condition and an operation was advised. The patient would not consent to this but sought further medical advice. Headache had shown no improvement. After much persuasion a blood Wassermann was obtained, which was negative, but a spinal Wasserman which was two plus. Patient was given arsphenamin and by the fourth treatment his headache had disappeared, and urine showed only a trace of albumin. Subsequently the postnasal discharge become less and less. From last reports the patient is still under observation and treatment.

This case well illustrates the complexity of some of our headaches and even though nasal pathology may be present, we must never close our eyes to the condition of the rest of the body.

DR. THOS. V. MURTO: All headaches are not due to nasal conditions and it is well for us to bear this fact in mind. Headache is not a disease, but merely a symptom of disease or local irritation.

Dr. Hombach in his splendid essay has pointed out how nasal conditions may produce headaches. No one disputes the fact that the frank case of acute sinusitis will give rise to characteristic headache. However, in mild or possibly chronic cases of sinus disease in which the patient has the associated condition

of either gout, anemia, diabetes or Bright's disease, we must remember that the associated condition may be the causative factor of the headache, as much as the sinus. Therefore, to avoid error, we should take careful histories and make or have made, in addition to a thorough nose and throat and ear examination, a complete physical examination, together with blood pressure, urine and blood examination.

Too often the nose and throat specialist fails to look at the eyes. He need not be an ophthalmologist to note inequality of pupils and congestion of the eyeball. As we know, glaucoma, iritis and uncorrected errors of refraction may give rise to headache similar to that caused by sinus disease.

True headache is due to pressure on, or stretching of, the dura mater. Thus anything that causes abnormalities of intracranial pressure, especially arterial hyperemia, as in renal disease and suppressed menstruation, and venous hyperemia, as in severe cardiac disease or emphysema, will cause headache.

Or, again, anything that gives rise to toxins which alter the intracranial circulation in the direction of increased blood pressure will cause headache. In this connection, I may mention the headaches of infective diseases, the frontal headache of hyspepsia (the morning headache after the alcoholic night before); the occipital headache of constipation, the headache from working in ill-ventilated offices and factories.

Also under the heading of increased intracranial pressure I need only mention intracranial tumors, gumma and abscesses, all of which at first may give rise to but slight indefinite headache.

And finally, a certain group of headaches may be due to peripheral irritation in various parts of the body. The explanation of this is that the trigeminal nerve, together with the first and second cervical constitutes the somatic distribution of that great complex of segments whose visceral distribution is the vagoglossopharyngeal, supplying the hearts, lungs, esophagus and upper part of intestine. The irritation of the vasoglossopharyngeal in any part may give rise to headache—as witness the frontal headache in some people on quickly swallowing ice.

These are merely the highlights on headaches from causes other than nasal which may confuse us and they by no means constitute all.

I wish to compliment Dr. Hombach on his paper, which he has excellently prepared and delivered, and voice my pleasure in discussing it.

DR. S. R. SKILLERN, JR.: Dr. Hombach's paper is a most interesting one, which gives us all much food for thought.

Is any complete physical examination worthy of its name without the opinion of a good sinuologist? No physician, unless he has had some special training in otolaryngology, is capable of passing judgment on the sinuses.

The doctor's presentation was "Headaches of Nasal Origin". This is often one of the late symptoms of sinusitis. In fact, in many cases, this symptom is absent, since headaches are one of the most inconstant of the many manifestations of sinusitis; discharge and general disability usually predominate.

When I speak of discharge, I do not necessarily mean a heavy, pustular one, since some of our worst cases do not show any signs of pus.

Dr. Hombach has mentioned "vacuum sinus headaches" as first brought to our attention by Dr. Sluder.

Personally, gentlemen (and I say personally since I do not know that all the members of our clinic agree with me), I do not believe there is such a phenomena. It is, in my opinion, against normal physiology, even pathology, if you will, to have the hemoglobin and other gas-carrying elements of the blood take up the oxygen from the sinuses and leave a vacuum.

In the first place, "Nature abhors a vacuum", so that I cannot see why the sinuses should prove an exception, when a vacuum is never heard of in any other part of the body.

The blood is essentially the carrier of two main gasses; namely, oxygen and carbon dioxid. It gives up one only in exchange for the other. The lungs are specialized tissue for the interchange of oxygen for carbon dioxid. The remainder of the body cells have the reverse action, in that they give up their carbon dioxid (for which the hemoglobin has a greater affinity) for oxygen.

Has anyone ever shown that the ciliated columnar epithelium of the sinuses has the same ability as the lung tissue? Not so far as I have been able to ascertain.

Granted that this lining membrane had such a function, would we have vacuum headaches? Certainly not! It would be a carbon dioxid headache.

You may argue that the bloodstream has the ability to take up gases, as shown by the rapid absorption of our artificial pneumothorax, or the absorption of nitrogen injected into the abdominal cavity, or the absorption of air beneath the superficial tissues. My answer is, certainly they should be absorbed, since they are foreign substances within this tissue. But the sinuses are normal air-containing cavities.

What was Sluder's thought in bringing forth his theory of vacuum sinus headaches? My understanding is that the swelling of the mucuous membrane about the ostium cuts off the aeration of the sinus, and that the air within the sinus is absorbed by the bloodstream, thus leaving a vacuum.

As soon as aeration is re-established, the pain from this vacuum disappears.

Isn't it a great deal more probable that the swelling of the mucous membranes around the ostium cuts off the blood supply, especially the venous return? If sufficient pressure is exerted to interfere with the venous return, we get a passive hyperemia with congestion and swelling of all the tissues.

It is possible for a leakage to occur from these vessels, which of course would be retained within the sinus; so that you would not only have pain from the engorged tissue, but also from the pressure of the escaped fluids on the highly sensitive membrane. The swelling from the engorged tissue may bring the two sides of the lining membrane into apposition. This would mean more pain. Now, if this pressure is suddenly relieved by shrinking the mucous membrane about the ostium, and the normal circulation is re-established, which in turn would relieve the swelling and congestion, wouldn't the pain disappear just as rapidly?

Sluder based his theory on the rapid relief of the pain without the appearance of any secretion. This would hold equally as true in acute congestion. If there had been any leakage from the vessels, its escape at the time of shrinkage could easily be mistaken for normal mucus or an excess of the cocain solution.

Consequently, I believe that the term "vacuum frontal sinusitis" is a "misname", and that this type of pain should be termed "congestive sinusitis".

The Present Status of the Lothrop-Skillern Radical Operation on the Frontal Sinus, with Especial Reference to Technique. Dr. Harry B. Goodspeed.

Appears in full in this issue.

DISCUSSION.

DR. CARLETON W. BULLARD: In choosing a technique for a radical frontal operation there are a few general factors which ought to guide in making a choice. One of these requisites should be whether or not it will allow the removal of the diseased mucosa as completely as possible, and a thorough exploration of the sinus cavity. Another factor of importance is whether it will allow the tissues to return to as normal as possible after operation. And then, too, there is the very important problem of facial disfigurement. This is more important than most of us realize, because the patient soon forgets the great relief from his suffering and discomfort that you have given him by operation, but is very slow to forget any disfigurement that may follow.

Dr. Goodspeed has already told you of these and many other advantages in the Skillern modification of the Lothrop radical operation, but I wish to emphasize the points which I have just stated, for these have been brought out by Dr. Skillern in a technique that overcomes the possibility of a poor result more than any technique before presented. This method allows a free view of the sinus cavity, and the tissue destruction is not great enough to cause failure of the sinus regaining its normal function and draining through an opening which does return within a short time after operation to a size

permitting permanent drainage, yet not large enough to act as an avenue of easy reinfection.

Other methods of radical frontal operations have given a poor cosmetic result, evidenced by the sinking in of the tissues of the supraorbital region. This is an extensive deformity. Dr. Skillern in his technique has nearly eliminated this possibility by separate skin and periosteal flaps. The incisions for these flaps are not made directly in line with each other, so that in suturing there is an overlapping of incisions, thus giving strength to the tissues over the operative bone cavity. I have seen and assisted in many of these operations. I have seen no untoward complications and the cosmetic result is splendid. After the eyebrow has grown in again to its normal length it is very difficult to see the line of incision made through it. There has been no sinking in of the tissues at any point.

I point out this to show that this technique has removed one of the greatest fears of radical frontal surgery. Every point has been most thoroughly covered in Dr. Goodspeed's paper showing why this is the operation of preference in the Graduate Hospital and of leading sinuologosists elsewhere.

DR. WM. O. SMITH: I believe I would rather yield the floor to Dr. Skillern and hear the one who has taken away the terrors of this operation.

DR. R. H. SKILLERN: I was very much gratified and rather pleased when I listened to Dr. Goodspeed's presentation of this operation. I do not want to flatter the boy, but I say with perfect frankness that many times have I listened to presentations which did not begin to compare with the one that he gave us tonight. It showed me that he not only understood what he was presenting but that he had also become firmly versed in the subject itself. He has taken up his work on the frontal sinus and he has mastered it and he has gone about it in the right way. He has gotten the theory if not the practice. He used pictures and every time a certain one came on the screen he was perfectly at home as to what it represented and what it meant.

Now, regarding the operation itself. This operation that we are now doing is a culmination of all of the operations that we did before. After the Killian comes this operation. This operation, as Dr. Goodspeed has said, takes about half the time of the Killian. We have not had a death in about 150 cases and we have not had any ill-timed results, such as blindness, paralysis, etc., so that, to my mind, where it is possible, it is the most preferable. If you have an infection of the bone, you have to resect the fistula and then you are back to the Killian. When we have a radical operation, it is by far the operation of choice. It leaves the terrors of the old frontal operation behind. With the old one, the patient made his will and bid goodbye to everybody. Nowadays, one goes in and expects to leave the hospital in seven to nine days a well man.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Chicago, Oct. 27, 1930, during the session of the American Academy of Ophthalmology and Oto-Laryngology.

One hundred and eleven candidates were examined, of which eighteen were either conditioned or failed.

The Board will hold an examination in Los Angeles in the spring, preceding the meeting of the Pacific Coast Oto-Ophthalmological Society, and in Philadelphia at the time of the meeting of the American Medical Association. An examination will also be given in the fall ,at Indianapolis, preceding the meeting of the American Academy of Ophthalmology and Oto-Laryngology, which will be held at French Lick Springs.

Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for proper application blanks.

DR. W. P. WHERRY, *Secretary.* DR. H. P. MOSHER, *President.*

IN MEMORIAM

ROSS HALL SKILLERN, M.D.

We chronicle with sorrow the passing of Dr. Ross Hall Skillern, of Philadelphia, who died in his office of a cerebral hemorrhage, Sept. 21, 1930, at the age of fifty-four.

A native of Philadelphia, his early education was received at Martin's School, in West Philadelphia, and the School of Biology of the University of Pennsylvania. He entered the Medical School of the University of Pennsylvania in 1893 and after graduation practiced with his father, the late Samuel Ruff Skillern, beginning post-graduate work in rhinology in Vienna and Gottingen, in 1906.

He was Professor of Laryngology in the Medico-Chirurgical College, 1913-1919, and Professor of Laryngology in the Post-Graduate Medical School of the University of Pennsylvania from 1919 until his death.

In the World War Dr. Skillern was commanding officer in charge of the Division of Head Surgery of the Base Hospital at Camp Sheridan, Alabama, later commanding Base Hospital No. 89 in France. He held the rank of Lieutenant Colonel.

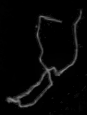
His textbook, "The Catarrhal and Suppurative Diseases of the Accessory Sinuses of the Nose," has received recognition and endorsement as one of the most popular of American textbooks in rhinology and is now in its fifth edition.

He contributed extensively to rhino-laryngologic literature and his writings are uniformly characterized by independence of thought and frankness of expression, and evidence of anatomical and technical mastery of his subject.

His research and special study has been devoted largely to the accessory sinuses of the nose.

He was a valued Fellow of the various national Rhino-Laryngological Associations and held executive offices as follows: President, American Academy of Ophthalmology and Oto-Laryngology, 1927; Chairman of the Section on Laryngology, American Medical Association, 1921; Vice-President, American Laryngological Association, 1925; President, American Rhinological, Laryngological and Otological Society, 1929-1930.

He was a member of the National Board of Examiners in Oto-Laryngology and rendered valuable services in this group.

He was an indefatigable worker, an enthusiastic and honest scientist, an excellent and natural teacher in laryngology; generous, frank, fearless and loyal. He was popular with his colleagues and always ready to co-operate in any movement or in any activity to promote the interests or progress of laryngology.

We extend our sincere sympathy and condolence to his family and offer this tribute to his memory. M. A. G.